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THE OCCURRENCE OF CONVULSIONS IN A SERIES OF OVER SEVEN HUNDRED VERIFIED INTRACRANIAL TUMORS

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IN studying the large amount of literature on convulsions, one is impressed by the great variety of etiologic factors discussed. On the other hand, in going over the records of a large number of cases of verified brain tumors, one finds that these patients frequently have convulsions as an early and major symptom. The purpose, therefore, of this review is to show that in any adult patient who has convulsions, the presence of a brain tumor must be given first consideration as the etiologic factor, for in no other condition may delay in diagnosis be so serious. The discussion of convulsions is confined exclusively to the preoperative period, but a complete tabulation of the cases in our clinic will form the basis for subsequent communications.

In this study, the records of 724 patients with verified brain tumors have been examined. Of these 724 tumors, 397 were cerebral or supratentorial tumors; 247 were cerebellar or infratentorial tumors; and 79 were pituitary tumors. Of this total 724 patients, 150 or 20.7 per cent had convulsions.

Of this group of 150 patients who had convulsions, 138 had cerebral or supratentorial tumors. Twelve of the cerebellar tumors had convulsions at some time during their course, but in none of the pituitary tumors did convulsions occur. This fact is readily understandable, for in cerebellar tumors convulsions are usually the result of the tremendous increase in intracranial pressure, and they almost without exception occur late in the course of the disease. On the

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TABLE I

1.	Verified brain tumors	724
	Patients having convulsions	150
	Percentage	20.7
2.	Verified cerebral tumors	397
	Patients having convulsions	138
	Percentage	34.8
3.	Verified cerebellar tumors	247
	Patients having convulsions	12
	Percentage	4.8
4.	Verified pituitary tumors	79
	Patients having convulsions	0
	Percentage	0

other hand, with rare exceptions, pituitary tumors, being extradural, do not produce an increase in intracranial pressure, so convulsions occur with extreme rarity.

Recent improvements in neurosurgical technic, added methods of tumor localization, and careful histopathologic studies of tumor types, have produced marked improvement in the prognosis of brain tumor cases. In spite of these improvements, however, the fact remains that early diagnosis is perhaps the greatest factor in producing a cure, for, after all, if a benign tumor is completely removed after the patient has become permanently blind, or suffered permanent impairment of certain areas of cerebral cortex, the results are much less gratifying. For that reason, an analysis of the relation of the time of onset of convulsions to the final diagnosis of tumor is of importance.

Twenty-seven patients in this series had had convulsions for periods of time ranging from five to twenty years before the diagnosis of tumor was finally made.

TABLE 2

TUMOR TYPES IN PATIENTS WHO HAD CONVULSIONS FROM FIVE TO TWENTY YEARS:

Meningeal fibroblastoma	8
Astrocytoma	8
Oligodendroglioma	2
Ependymoma	2
Neuro-epithelioma	1
Glioma, unclassified	1
Suprasellar cyst	1
Angioma of cortex	1
Sarcoma	1
Cyst, unclassified	1
Tumor, unclassified	1

Needless to say, these patients had benign, slowly growing tumors, but the fact also remains that even a benign tumor can, in from five to twenty years, attain such enormous size that marked cortical degeneration will occur, and its removal becomes, because of its size and increased vascularity, technically much more difficult. The following case is an excellent illustration of this point.

CASE 1. Mrs. O. E. A., aged 51, was admitted to the neurosurgical service on Nov. 20, 1934, with a chief complaint of convulsions. The family history was unimportant and no member of her family had suffered with epilepsy. Since birth Mrs. A. had had an internal strabismus.

Present Illness: More than ten years before admission she had begun having convulsions which always started in the toes of her right foot and spread up the

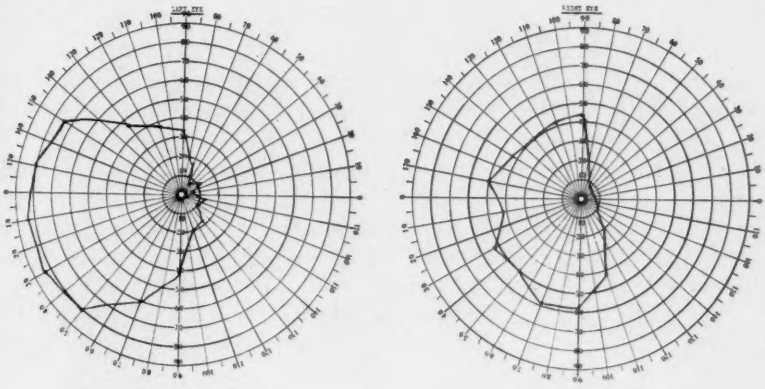

Fig. 1. Right homonymous hemianopsia as a result of pressure on the left occipital lobe. (Case 1)

leg to the muscles of the thigh. These attacks were never accompanied by loss of consciousness, and they always remained Jacksonian in type. They recurred at intervals of a few days to a few weeks without progression for over six years. She then had an attack which spread to the right arm and the right side of the face. Following this convulsion she was unable to speak for twelve hours, but then her speech gradually returned to normal. In spite of phenobarbital and bromides, she continued to have right-sided convulsions more and more frequently, and for two years her family noticed a progressive failure of memory and mental dulness. This was attributed, by the many physicians whom she consulted, to the mental deterioration which occurs during the course of epilepsy. During the year before admission she had developed a progressive right hemiparesis, but there was never any headache or vomiting.

Neurologic examination, when we saw her, revealed bilateral choked discs of long standing with a recent hemorrhage in the right eye. There was a left internal strabismus. There was a right hemiparesis, involving the face, arm,

and leg, but most pronounced in the leg, with pathologic reflexes on the right side. In addition she had an almost complete right hemonymous hemianopsia (Fig. 1). X-ray showed destruction of the dorsum sellae with some slight calcification in the postcentral area, and thickening of the parietal bone (Fig. 2).

The diagnosis was a slowly growing tumor, most probably a meningeal fibroblastoma, extending parasagittally from the motor cortex on the left side back to the left occipital lobe.

Operation: On November 23, a large left cerebral flap, extending to the median line, was reflected. Because of the increased vascularity of the bone and dura, the bleeding was so terrific that it was necessary to give a transfusion.

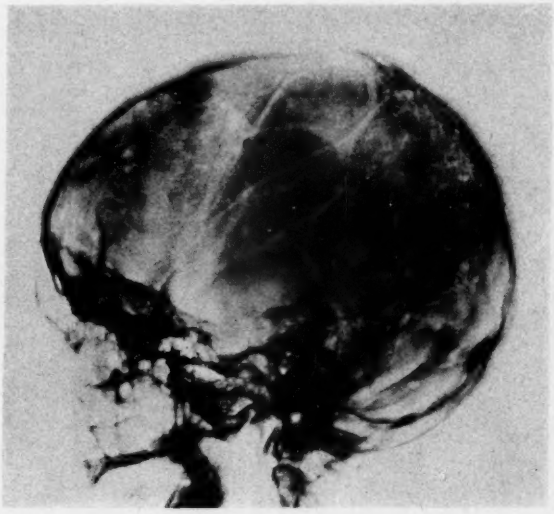

Fig. 2. Destruction of the dorsum sella and thinning of the parietal bone as a result of the prolonged pressure from the slowly growing tumor. (Case 1)

A large piece of muscle, taken from the patient's thigh, was used to stop the bleeding from the region of the longitudinal sinus, and the flap was replaced, for it was out of the question to complete the operation in one stage.

On the 26th, the second stage was done. A very large parasagittal meningeal fibroblastoma was exposed, and all except a small amount of tumor in the occipital region was removed by coring it out with the electric knife. The tumor weighed 205 Gm. It was necessary, because of a fall in blood pressure, to stop when this much had been accomplished. On December 27, the removal of the tumor was completed. The small piece which was taken out at this time weighed only 12 Gm.

She was discharged on Jan. 20, 1935. At that time she still had her right hemiparesis. Her fundi were normal, and the mental state was markedly improved. The visual fields, however, were unchanged.

Comment: Because of delay in diagnosis, this patient had to undergo three operations instead of one. Her hospital stay was sixty days instead of the usual twelve to sixteen, and the right hemiparesis, which would have cleared up promptly had operation been done sooner, will remain permanently. Fortunately, her visual acuity was not seriously impaired, but the homonymous hemianopsia will remain as a result of the permanent cortical damage in the occipital lobe.

Astrocytomas

In studying the relation of convulsions to the various types of cerebral tumors in this series, some interesting facts are brought out. Quite naturally, it is found that the highest incidence of convulsions is in the slowly growing types of tumors. The astrocytomas produce convulsions most frequently.

Table 3

Astrocytomas, Verified, Cerebral

Verified astrocytomas, cerebral	51
Patients having convulsions	30
Percentage	60

Location of tumor as related to convulsions:

	Number	*Percentage*
Frontal lobes	21	70
Temporal lobes	3	10
Parietal lobes	6	20
Occipital lobes	0	0
Cerebral astrocytomas in which convulsion was first symptom		26
Percentage of total number (26 of 51)		52
Percentage of those having convulsions (22 of 26)		85

Length of time convulsions present before other symptoms:

	Number	*Percentage*
Less than 3 months	4	15.50
3-6 months	5	18.75
6-12 months	1	4.00
1-2 years	5	18.75
2-3 years	3	12.00
3-5 years	0	0.00
5-10 years	6	23.00
10-15 years	2	8.00
15-20 years	0	0.00
Total	26	100.00

Type of convulsion	*Number*	*Percentage*
Generalized	11	37
Jacksonian	14	45
Generalized and Jacksonian	5	18

Of 51 cerebral tumors in this pathologic group, 30 cases, or 60 per cent, had convulsions. In 26 of the 30 cases, or 52 per cent of the total series, *a convulsion was the first symptom*. Since these tumors are frequently cystic and are permanently cured by the removal of the growing portion of the tumor in the cyst wall, it is a great tragedy when permanent damage occurs to the optic nerves before a cure is effected. This is well illustrated by the following case:

CASE 2. W. P., Aged 19, was admitted to the neurosurgical service on Feb. 16, 1934, on account of convulsions, blindness and deafness.

Present Illness: This had begun four years before admission when suddenly and without warning he had a generalized convulsion with loss of conscious-

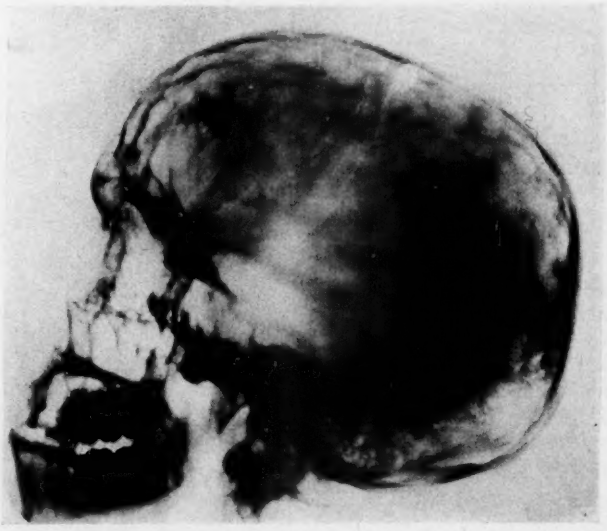

Fig. 3. Extreme degree of convolutional atrophy with almost complete destruction of the sella as a result of the prolonged intracranial pressure. (Case 2)

ness. The attack was typically epileptiform. He had continued to have similar attacks at frequent intervals, always without aura; because of the resultant embarrassment, he had stopped school. Early in 1933 he had started having severe headaches, chiefly frontal and parietal in location. These headaches increased in severity, so that for about five months prior to admission he had been bedridden. Three months before he came in, he had noticed that his vision and hearing were rapidly failing, and at the time of admission he was totally blind and apparently totally deaf. He was extremely uncooperative. Because of the loss of vision and hearing it was impossible to communicate with him in any way, and his condition was more like that of an animal than a human being. He was totally incontinent, and apparently had lost both the senses of taste and smell, for no matter what was put in his mouth he chewed and

swallowed it, and he did not react in any way to strong and offensive odors.

There were numerous manifestations of Von Recklinghausen's disease: pigmented spots, fibromatous nodules, and large lymphangiomatous masses on the back and the left ankle joint. Some of these had been present since birth.

Neurologic Examination: An apparent total loss of smell was present. The pupils were fixed and dilated and there was total loss of vision. The fundi showed marked papilledema with hemorrhages, and extreme secondary optic

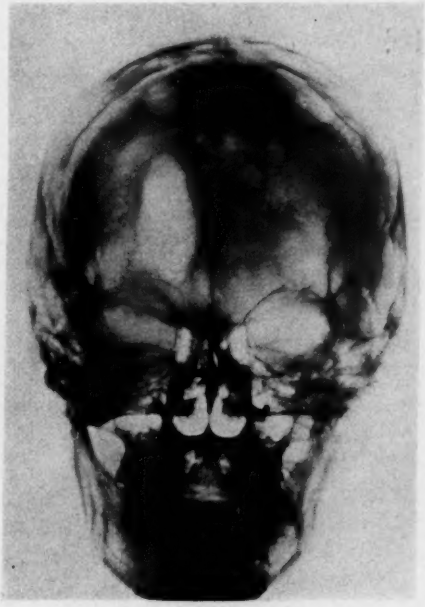

Fig. 4. Dilatation of the left ventricle with failure of the air to go across into the right ventricle. (Case 2)

atrophy. His hearing seemed entirely gone, and there was no reaction when bitter substances were placed in his mouth. There was a peculiar fine tremor of the left hand, and bilateral pathologic reflexes.

X-rays of the skull showed marked pressure with a large deep sella, the floor of which was eroded, and the dorsum pushed forward (Fig. 3).

The diagnosis was brain tumor, probably in one of the frontal lobes, but ventriculography was necessary for accurate localization. This procedure was carried out on February 21. The plates were interpreted as follows: "The left ventricle is dilated and no air goes across into the right ventricle (Fig. 4). However, when air is put into the right ventricle, the posterior horn appears normal but the anterior horn and body are almost obliterated and are pushed down and slightly across the median line (Fig. 5). There is a tumor in the right frontal lobe which has almost obliterated the foramen of Munro."

On February 24, a right frontal flap was reflected. A huge cyst was evacuated and incised and the nubbin of tumor in the cyst wall was entirely removed.

His postoperative recovery was rapid. On the second day he could hear low tones and would answer questions. At the present time, except for high tones his hearing is normal. He is a bright, intelligent young man, neat in his dress and meticulous as to his personal habits. He has progressed rapidly in his knowledge of Braille, for his secondary optic atrophy is permanent and complete, and his vision is totally lost.

Fig. 5. Air put into the right ventricle shows normal posterior horn with almost complete obliteration of the body and anterior horn and displacement of the body and anterior horn across the median line indicating tumor in the right frontal region. (Case 2)

Comment: This case is an excellent surgical result, but pathetic when one considers that 3 or 4 months' delay meant the difference between total blindness and good vision. Had encephalography been done before any of the signs of pressure appeared the diagnosis would have been obvious, and the operative procedure less formidable. The deafness is very unusual with this type of tumor, and must have been due to the increased intracranial pressure, for it cleared up promptly after the pressure was relieved.

Meningeal Fibroblastomas

Meningeal fibroblastomas, like the astrocytomas, frequently produce convulsions.

Table 4

Meningeal Fibroblastomas, Verified, Cerebral

Verified meningeal fibroblastomas, cerebral	79
Patients having convulsions	28
Percentage	35.4

Location of tumor as related to convulsions:

	Number	*Percentage*
Frontal lobes	22	78.5
Temporal lobes	3	11.0
Parietal lobes	2	7.0
Occipital lobes	1	3.5
Cerebral meningeal fibroblastomas in which convulsion was first symptom		22
Percentage of total number (22 of 79)		28
Percentage of those having convulsions (22 of 28)		79

Length of time convulsions present before other symptoms:

	Number	*Percentage*
3-6 months	4	18
6-12 months	2	9
1-2 years	4	18
2-3 years	3	14
3-5 years	1	4
5-10 years	3	14
10-15 years	3	14
15-20 years	2	9

Type of convulsion	*Number*	*Percentage*
Generalized	10	36
Jacksonian	15	54
Generalized and Jacksonian	3	10

There were 79 verified cerebral tumors of this classification, and 28, or 35.4 per cent, of these had convulsions. Twenty-two of the 28 had convulsions as the first symptom, and in nine of these, the convulsions occurred from five to twenty years before other symptoms developed. In this group, as in the astrocytomas, frontal lobe tumors produced convulsions much more frequently than those located in other regions.

Spongioblastoma Multiforme

Spongioblastoma multiforme frequently produce convulsions, but they occur much less frequently as the first symptom.

Table 5

Spongioblastoma Multiforme, Verified, Cerebral

Verified spongioblastoma multiforme, cerebral		66
Patients having convulsions		25
Percentage		38
Location of tumor as related to convulsions:		
	Number	*Percentage*
Frontal lobes	10	40
Temporal lobes	12	48
Parietal lobes	3	12
Occipital lobes	0	0
Cerebral spongioblastoma multiforme in which convulsion was first symptom		12
Percentage of total number (12 of 66)		18.5
Percentage of those having convulsions (12 of 25)		48
Length of time convulsion present before other symptoms:		
	Number	*Percentage*
Less than three months	6	50
3-6 months	1	8
6-12 months	3	25
1-2 years	2	17
Type of convulsion	*Number*	*Percentage*
Generalized	5	20
Jacksonian	18	72
Generalized and Jacksonian	2	8

Of the 66 tumors of this classification, 25 or 38 per cent had convulsions, but in only 12 of these did they occur first, and in the majority of these cases the attack was followed immediately by other symptoms,—usually by the signs of increasing intracranial pressure. This is the only group in which convulsions occurred less commonly in the frontal lobes, but due to the infiltrating nature of the tumor, it is frequently impossible to determine definitely its point of primary origin. In only 2 of the 66 cases were convulsions present longer than a year, and in none for more than two years.

Other Cerebral Tumors

Examination of the other tumor types shows nothing of particular interest. The principles brought out above still hold, i. e., in

TABLE 6

OTHER CEREBRAL TUMORS, VERIFIED

Type	*Number*	*Number Having Convulsions*	*Percentage*	*Number Having Convulsions As First Symptom*	*Percentage*
Astroblastoma	10	4	40	2	20
Neuro-epithelioma	5	3	60	3	60
Medulloblastoma	3	1	33	1	33
Ependymoma	16	6	37	4	25
Spongioblastoma polare	12	2	17	1	8
Glioma unclassified	25	2	8	2	8
Cysts	28	4	14	2	7
Tumor unclassified	1	1	100	1	100
Ganglioneuroma	3	1	33	1	33
Cholesteatoma	1	0	0	0	0
Osteochondroma	1	0	0	0	0
Pinealoma	11	3	27	0	0
Oligodendroglioma	12	5	42	4	33
Angioma	13	8	62	6	45
Hemangioblastoma	7	2	28	2	28
Granuloma	21	5	19	1	5
Perineurial fibroblastoma	2	1	50	0	0
Miscellaneous and metastatic	40	8	20	6	15

the slowly growing types, such as oligodendroglioma and ependymoma, convulsions occur frequently, and may well be the initial symptom. In the rapidly growing tumors, such as the astroblastomas, the pinealomas, and the medulloblastomas, they occur fairly frequently, but usually late in the course and usually long after other tumor symptoms have appeared.

As the statistical tables show, convulsions occur most commonly in frontal lobe tumors, particularly with the slowly growing, favorable types. This fact is worthy of emphasis, for if tumors are diagnosed early, before permanent damage has been done to the motor cortex, the anterior portion of the frontal lobes, particularly the right, can be radically removed with no definite demonstrable loss of function.

From the standpoint of types of convulsions, Jacksonian, or localizing, attacks are more common than the generalized attacks. We have classified as Jacksonian any convulsion which had a definite localizing value, e.g., a motor convulsion which was confined to one part of the body or definitely and systematically started in one part. Convulsions which were preceded by an aura such as an hallucination of smell, taste, vision or hearing were also included in this

classification, but no attacks of unconsciousness in which convulsive movements were absent were considered as true convulsions. Some patients had both generalized and Jacksonian types of convulsions, as the following case will show.

CASE 3. Miss A. V. L., aged 27, referred by Dr. H. W. Wiese and Dr. A. D. Carr, was admitted to the neurosurgical service on Feb. 28, 1933.

Present Illness: Seventeen years before, at the age of 10, she had had a generalized convulsion. The convulsions had recurred at frequent intervals and were always epileptiform in character:—i.e., there was no aura, the convulsion was generalized, consciousness was lost, there was increased salivation, the tongue was bitten, and the attack was followed by a period of coma, usually about thirty minutes. In addition to the generalized convulsions, she had also had petit mal attacks for ten years, during which she would lose consciousness and stand with her eyes fixed and staring for a few seconds. Two years before admission, she went into status epilepticus. She had 31 convulsions before consciousness was regained.

Two days before admission, she again began having a series of attacks. Because of this condition, hospitalization was advised. In studying the convulsions which she had after admission, Dr. Carr and Dr. Wiese noticed that they started with a flexion of the left forearm on the arm, accompanied by a turning of the head to the left side. As soon as the convulsions had been controlled, X-ray plates were made, and these showed slight intracranial calcification in the right frontal region. Because of this, she was transferred to the neurosurgical service with a diagnosis of right frontal lobe tumor, either a cyst or a meningeal fibroblastoma.

On March 6, 1933, ventriculography was done. The films showed that the anterior horn of the right ventricle was pushed backward, so a right frontal flap was immediately reflected. A very hard, irregular tumor mass was removed together with the surrounding cortex, and the flap replaced without leaving a decompression. She was discharged on March 20, and with the exception of an occasional mild petit mal attack, usually about the time of menstruation, she has remained well.

Comment: Though this case had been studied by competent observers, it is well established that only after 17 years did the convulsions become Jacksonian in type. It is unusual that she developed no signs of increased intracranial pressure, but the lack of localizing signs is easily explained by the location of the tumor.

The pathologic type of this tumor has never been established, although many pathologists have been given an opportunity of studying it.

CEREBELLAR TUMORS

As was previously stated, convulsions do not occur frequently in cerebellar tumors.

TABLE 7

CEREBELLAR TUMORS, VERIFIED

Verified cerebellar tumors			247
Patients having convulsions			12
Percentage			4.8
Cerebellar tumors, verified, in which convulsion was first symptom			0
Type of convulsion:			
	Number	*Percentage of total number*	*Percentage of those having convulsions*
Generalized	6	2.4	50
Jacksonian	1	.40	80
Cerebellar fits	5	2.0	42

Only 4.8 per cent of this series of 247 had convulsions, and in no case was a convulsion the first symptom. Cerebellar fits occurred in only five cases, and one patient with a tumor of the right cerebellar lobe had definite Jacksonian convulsions involving only the right hand and arm.

CONCLUSIONS

1. Since convulsions are so frequently due to brain tumors, every adult having a convulsion should be considered to have a tumor until proven otherwise.

2. Convulsions occur much more commonly in cerebral tumors than in cerebellar tumors.

3. No patient in this series with a true pituitary tumor had convulsions.

4. In the slowly growing and surgically favorable types of tumors, such as the astrocytomas and meningeal fibroblastomas, convulsions occur frequently, and are, in many cases, the first symptom of tumor.

5. Frontal lobe tumors produce convulsions more frequently than those in the other cerebral lobes.

6. In this series, 27 patients had convulsions for from five to twenty years before the appearance of other symptoms of tumor.

7. One of the most necessary requisites for the successful surgical treatment of brain tumors is early diagnosis, for if the diagnosis is delayed, serious and permanent damage to the optic nerves and to the cortex may occur. For this reason, it is necessary to rule out the presence of a brain tumor in any patient who has convulsions.

WHAT CAN WE EXPECT FROM RADICAL SURGERY FOR RECTAL AND RECTOSIGMIODAL CANCER?

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THE assertion that rectal cancer remains local for a long time and metastasizes slowly, and therefore offers a favorable prognosis has been made so often that it is almost trite to repeat it. Nevertheless, it is easily proved statistically that carcinoma of the rectum for a number of reasons is of hopeful outlook if attacked radically by surgery before it has gone beyond its primary local stage.

The diagnosis of carcinoma of the rectum may be made in every case by digital or proctoscopic examination, and surely no general physical survey is complete which does not include both of these examinations. There are no pathognomonic early symptoms of cancer of the rectum or rectosigmoid of which I am aware. Probably blood in the stool or on the stool is the one symptom which sends more people to a physician for examination than any other, but this symptom is the result of secondary changes in the tumor itself, ulceration having progressed to such a point that the mucosa is destroyed and the blood vessels are opened into.

That the cancer has been present a long time before such changes take place in the usual course of events is a reasonable supposition. The mere presence of a tumor in its earliest stages rarely produces symptoms until it has grown to such a size that it either produces bowel irritability and a consequent deviation from the normal bowel habit, or encroaches on the lumen producing some form of intestinal obstruction, ranging from mild to complete.

Cancers of the rectum are situated most often high in the rectum at its juncture with the sigmoid colon, less frequently in the ampullary segment of the bowel, and only occasionally low down toward or in the anal canal. The presence of a tumor in the latter location results in early symptoms due to irritation of the rectal sphincter and a consequent tenesmus. Tumors in the rectosigmoid are most prone to produce obstructive phenomena and occasionally manifest themselves as an acute intestinal obstruction without warning.

Adequate digital and proctoscopic examination of the rectum will in every case reveal the presence or absence of a lesion. Usually the lesion is unquestionably identifiable on inspection, but if there is a question, or if for other reasons one wishes to do a biopsy,

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microscopic section ratifies or eliminates the question of malignancy. The use of the opaque medium and x-ray examination for tumors of the rectum and rectosigmoid is not a satisfactory procedure because of the overlying coils of bowel in the pelvis which interfere with accurate visualization. Moreover, it is unnecessary if digital or proctoscopic examination reveal an organic lesion.

We possess two offensives against malignancy of the lower gastrointestinal tract, namely, surgery and radium. The exact role of radium in the treatment of cancer of the rectum and rectosigmoid is uncertain at present. That it has a definite place and probably will be a more important factor in the future seems reasonable, but at present one does not know which tumor is radio-sensitive and unquestionably the control of metastases by radium is uncertain. That some tumors can be made to disappear by the direct application of radium around the base and over the surface of the tumor is well recognized, but once the cancer cells have migrated, their destruction by radium is inconceivable. Furthermore, we know that actually 46 per cent of cancers of the rectum have metastasized to the regional lymphatics at the time the resection is performed, and consequently to employ routinely an agent which cannot affect these distant deposits seems illogical in the light of more satisfactory results following radical extirpation.

That there will be a field of cooperative management of radium and surgery is a distinct hope and not an unreasonable expectation, but it remains for the future to develop not only better technical methods of application of irradiation, but its availability in operable cases both before and after surgery is undertaken. The role of radium as a palliative therapeutic agent is still debatable, I think.

Surgery applied radically gives a most hopeful prognosis to sufferers with cancer of the rectum and rectosigmoid following the same surgical principles that Halstead established for the radical cure of cancer of the breast, or that are attempted for cancer of the stomach, lip, or other organs.

By reason of the recently developed adequate preliminary measures plus accurately thought out and judicially applied technical maneuvers, the surgical attack on malignancy of the lower bowel may be indulged in with a high operability, a reasonable mortality figure, and a high percentage of five-year cures. That there is no one standard operation which may be applied to all cases of cancer of the rectum and rectosigmoid with a hope of a reasonably high percentage of five-year cures is unquestionably true. However, there are two types of maneuver, one of them radical, and done either in two stages or one stage, namely Miles' one-stage operation,

or Rankin's or Jones' two-stage operation; and the other, Mummery's posterior resection following colostomy which permits a less radical gland dissection. Either of these will cure according to the activity of the growth, its extent, and the presence or absence of metastases in from 38 to 72 per cent of all resectable cases.

The thesis that colostomy is an essential part of any operation which is a radical extirpative procedure, expresses, I think, the feeling of the vast majority of experienced surgeons who operate upon malignancies in this location. While a few patients may live over the five-year period when operated upon by segmental resection or local removal of the growth or fulguration of the growth, or some other operation of the type which has as its main objective the saving of the sphincter mechanism, when one views the end—results from the standpoint of 100 or 500 operative cases, it is clearly demonstrated that radical surgery is well worth while, and radical surgery may not be applied save only by the use of a colostomy.

Colostomy is a procedure much maligned by the medical profession and a horror when first broached to a layman who needs one, but with proper manufacture, adequate care, and the development of a proper attitude by the patient toward the colostomy, it is a bearable burden which does not too greatly handicap anyone from his social or professional duties.

The ideal operation for cancer of the rectum, I think, is agreed to be the one-stage combined abdominoperineal resection of Miles. This cannot be applied to all cases but increasing experience, better preoperative preparation, and more meticulous attention to technical details, has enabled me to utilize this operation now much more frequently than formerly. I modified Miles' operation, performing it in two stages for a number of years with satisfactory operative mortality and I believe satisfactory end-results, but during the past year I find that I have done the one-stage operation almost to the exclusion of my own two-stage procedure where a radical operation could be performed at all. That there are some cases which must continue to be done by the colostomy and posterior resection of Mummery, is beyond question in my opinion, and one must always have this procedure in reserve.

Probably a surgeon who does only a few operations each year for cancer of the rectum will still be inclined to employ this less radical operation because of its greater ease of performance and lower operative mortality, but the end-results will be disappointing in comparison with the five-year cures of the one-stage or the graded radical resection, the operability will be no higher and the operative mortality in experienced hands will be but little different.

No cancer of the rectum except one which is producing complete intestinal obstruction should, I believe, ever be operated upon without preliminary measures to decompress and cleanse the bowel, while at the same time rehabilitory measures are employed to increase the individual's general resistance. Decompression is accomplished by irrigations and mild purgation, while the diet of the patient consists of non-residue foods, and his general resistance is built up by feeding large amounts of carbohydrates.

Technic

The types of radical extirpative procedures with which I have had the most experience have been, first, the graded maneuver which I described in The American Journal of Surgery (February, 1935) and which is in reality a modification of Miles' one-stage operation; and second, Miles' one-stage combined abdominoperineal procedure.

In selecting an operation for an individual case, the preliminary and exploratory stages are identical, regardless of whether one does a one-stage operation or a graded one. Exploration is made through a low midline incision and is uniformly carried out from above downward, exploring the content of the upper abdomen first and the growth last because of the question of infection in the perirectal tissues which may be loosened by even gentle manipulation and handling.

Having decided to do a combined abdominoperineal operation, the first decision is whether or not to do it in one or two stages and if the Miles technic is decided upon, the ligation of the inferior mesenteric vessels close to their origins is the first step. Having doubly ligated these vessels and tied off the proximal blood supply in the mesentery up to a selected point where the single-barreled colostomy is to be made, the bowel is clamped with the Zachary-Cope modification of the DeMartel clamp and divided with a cautery. The upper end which is subsequently to be brought out for a colostomy is now packed away and the subsequent stages of the operation are carried out in order.

Peritoneal flaps are manufactured on either side and the rectum is separated completely from the bladder and the peritoneal covering of the bladder is raised so that there will be adequate loose peritoneum to manufacture a new pelvic floor. Then the rectum and the perirectal tissues are mobilized from the hollow of the sacrum by hand dissection. The latter dissection is carried well over to the sides of the pelvis and the ligamental attachments of the rectum are severed with long scissors.

It is possible to make a very clean dissection of the pelvis, identify-

ing both ureters and the large blood vessels, sweeping out the fat of the perirectal tissues into which metastases would take place. At this point all of the bowel to be removed is tucked down into the pelvis and a new pelvic floor is made by closing the peritoneal flaps accurately and with two layers of sutures if catgut is used. This covers all raw surfaces and the colostomy is now effected by making a small stab wound mid-way between the anterior superior spine of the left ilium and the umbilicus, and bringing out the proximal bowel with the metal clamp on it.

Mr. Miles attaches this single barreled colostomy loosely to the skin after cutting away a sufficient amount of skin to insure a soft, not too tightly contracting wound from scar tissue. He also opens the bowel after having completed the rest of the operation. I have found no disadvantage in leaving this clamp on for 24 to 48 hours provided no distention develops.

Having completed the colostomy, the abdominal wound is closed in the routine manner, the patient turned on the side and the perineal part of the operation carried out quickly and easily. The anus is sutured with a purse-string suture and then encircled with two curved incisions which extend up to the sacrum. The coccyx is disarticulated and the lateral dissections are carried forward up to meet the peritoneum. The greater part of the levator ani muscle and the perirectal fat in the ischiorectal fossa is removed in this manner with the rectum and the major portion of the sigmoid and its mesentery.

The posterior wound and pelvis is irrigated with 1 to 1000 solution of bichloride of mercury and subsequently washed out with salt solution, then packed snugly with gauze between which and the raw surfaces is interposed a large sheet of rubber tissue. This latter step is a most important and helpful one not only to the surgeon who has to remove the packing subsequently, but to the patient as well. This packing is put in snugly and left in for a matter of 48 hours when one-half of it is removed. This allows exudate to form and to support the pelvic floor and at the end of 60 to 72 hours the rest of it is removed and a clean, granulating wound is found.

The drainage is easily maintained because one-half of the peritoneal wound is left open, but healing is remarkably quick in many of these cases and the average one will be completely scarred over in about six weeks. Irrigations are carried out as seem indicated. Routinely, during the past two years, I have followed the suggestion of Mr. A. Lawrence Abel, of London, doing a presacral resection in all of these cases in which I have done either a one- or a two-

stage combined abdominoperineal operation, and I am convinced that the bladder conditions following pelvic dissection and destruction of so many of the motor nerves, are much improved by this procedure.

The bladder seems to empty more readily and one can get away from prolonged catheterization in many of the cases. Unquestionably, bladder complications and sometimes serious renal sequelae do develop in many of these radical resections, but if one considers the amount of tissue that is removed and the obvious injury to so many of the pelvic nerves, there is small wonder that there is not more often a prolonged and unfortunate complication.

The same type of operation can be carried out in two stages by my own maneuver in cases where the formidableness of the operation in one stage prohibits its employment, and in other cases which for various reasons, it seems best to employ a two-stage operation. The exploration is carried out as in Miles' operation, but the manufacture of the colostomy is slightly different. A point in the bowel where the colostomy is to be made is selected and the blood supply to the point is ligated, taking care to preserve the arcs of the inferior mesenteric vessels, and ligating only the ascending branches which run from them up to the bowel over a sufficient distance to make the colostomy.

Here again, the Zachary-Cope clamp is used, but is taken off the distal end which is inverted with the running Lambert suture. Care is taken to be sure that there is adequate vascularization to both ends, then the proximal end is brought out through a stab wound just as in the one-stage operation, and the wound is closed. The patient is allowed to go for a number of weeks until the optimum improvement in his condition indicates that it is time for the second stage.

This second stage is carried out by a perineo-abdominal type of procedure, the dissection beginning behind and being carried up to the peritoneal reflection, the same tissues being removed as in the one-stage procedure. After reaching the peritoneum behind, the rectum is encased in a rubber glove, thrust into the wound, and the wound closed. The patient is then turned on his back, put in a deep Trendelenburg position and the abdomen opened through a low mid-line incision. Now, the inferior mesenteric vessels are ligated, the peritoneal flaps are manufactured identically as in the former procedure, and the whole segment of bowel lifted out through the abdominal wound.

The manufacture of a new pelvic floor is easily accomplished and the abdomen is closed in the routine manner. The patient is turned

on his side, the posterior wound is packed with gauze, and covered with rubber tissue. The postoperative steps are identical with those in the other type of procedure.

It is impossible to separate even approximately, operability, operative mortality, and five-year end-result statistics, for they are so inextricably bound up in each other that the same factors which influence favorably or unfavorably one, act in the same manner upon the other. It is my belief that the most important single factor which influences the prognosis of cancer of the lower gastrointestinal tract is the intrinsic activity of the cancer cells themselves as represented by their ability to differentiate or not differentiate from the normal. This influence is a direct one on metastases, both glandular and hepatic, and as Broders has shown, the rapidity of metastases is in direct ratio to the activity of the cell growth. Low grade malignancy remains local for a long time and on resection, the glands show no malignancy. Conversely, high grade malignancy remains local but a short while and local and distant metastases are evidenced in a high percentage of rectal cancers.

Modifying conditions which influence operability and therefore mortality and prognosis, are due to either complications arising from the malignancy, such as fixation, abscess formation, or perforation resulting in immobility, or some such general conditions of the host as co-existing diseases, obesity, extremes of age, duration and direction of the growth.

Standards of operability are difficult to define accurately, but obviously should be as elastic as possible to include borderline cases and thus extend the scope of the operative act to all cases which may be removed with a reasonable mortality rather than attempting to reduce the mortality figures, and at the same time reduce the operability rate.

During the past five years I have maintained an average operability of three out of four cases of cancer of the rectum and rectosigmoid (accurately, 76 per cent). Over this period of time the mortality has been reduced to about 8 per cent for the very radical operation of combined abdominoperineal resection in one and two stages, and the mortality for the less extensive but very useful operation of Mummery, has been maintained at approximately 5 per cent. I have now done 104 radical combined abdominoperineal resections in one or two stages with eight deaths—a mortality of 7.6 per cent.

In removing three out of four cancers of the rectum and rectosigmoid, I have been able to apply radical procedures to more than one-half (59.5 per cent) of the cases as they present themselves

for exploration, and the less radical operation of posterior resection following colostomy to the rest. I have no enthusiasm for, and am inclined to deprecate the more recently popularized procedure of operative attack on cancer of the rectum by electrocoagulation methods, in the belief that while they may control or destroy the local growth, much as radium does, they have no influence on metastases and one should remember that almost half of the resected specimens (46 per cent) show glandular involvement as having taken place at the time of operation.

Prognosis

Statistical estimates of the length of life following operations for cancer of the rectum and rectosigmoid show conclusively that the more radical operations of the Miles type invariably result in a larger number of cures than the less formidable but frequently essential procedures such as colostomy and posterior resection. Miles, with an operability of approximately 30 per cent, had 79 per cent five-year cures in a relatively large series of cases. Jones, with a higher operability and a similarly satisfactory mortality, had 50 per cent five-year cures following his graded operation. Any operation which is rewarded with a five-year cure of one out of two cases must be considered a most satisfactory procedure, particularly if this is accomplished without sacrifice of the operability curve.

A series of 300 cases of posterior resection following colostomy which I studied a number of years ago, yielded a five-year cure of only 34 per cent—a contrast which persuaded me that more radical procedures were demanded and could be satisfactorily carried out by the extension of preparatory measures and more meticulous care at the operative stage.

My own series of 104 cases of radical resection by the Miles, or the Rankin modification of Miles' technic, has not been done a sufficiently long time to allow computation of five-year results, but the overwhelming evidence from other series will, I am sure, be borne out in defense of the radical procedure being selected where it is at all compatible with hospital mortality and morbidity rate.

Despite the enormous technical advances of the past two decades and the increasingly hopeful prognosis percentages being frequently compiled, the most essential step, namely earlier diagnosis, is necessary to further progress in the treatment of cancer of this particular field. The present average duration of symptoms—eleven months before medical advice is sought, must be reduced enormously before great strides in longevity can be made.

RECENT DEVELOPMENTS IN SURGERY OF THE SYMPATHETIC NERVOUS SYSTEM

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THE past year has seen several applications of sympathetic surgery, not entirely new, but in which a more widespread interest has arisen, namely, in multiple sclerosis, essential hypertension and pelvic pain. Besides these, my own interests have concerned several other subjects, among them migraine, atypical pains of head and neck, the Parkinsonianism of encephalitis, and the pain of abdominal malignancy. It is only natural that in such a young and robust field advances and refinements of surgical technic should occupy considerable attention.

MULTIPLE SCLEROSIS

In 1933 Royle[1] reported four cases of multiple sclerosis treated by cervicodorsal trunk section with excellent results in all cases. In 1934 Wetherell reported one case[2] and in 1935 eight cases[3] treated by cervicodorsal sympathectomy with promising results. I have done fifteen cases.

The rationale of this procedure is based on the theory that the sclerosis which occurs in the central nervous system is due to interference of blood supply[4,5] and that sympathectomy gives its beneficial effects by increasing the blood supply to the brain and spinal cord. This theory is tenable if one considers the history of these cases. One of the most characteristic features of the disease is the occurrence of remissions during which the patient will experience definite improvement. If one assumes that the symptoms and findings of the disease appear only after the sclerotic areas have formed in the brain and spinal cord, then one has no explanation for the occurrence of remissions. On the supposition that a portion of the cord and brain has not been functioning due to a lack of blood supply, then the remissions may be explained rationally by a temporary increase of blood supply restoring function for the time being. The pathology of the disease may be areas of sclerosis which are functionless, surrounded by zones of tissue in which the cells are viable but are not functioning due to insufficient blood supply. As time goes on the sclerotic areas increase in size and the viable but functionless zones also radiate and increase in size.

If this theory is correct, it should be possible to save these func-

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tionless but viable zones by increasing the blood supply through removal of the necessary sympathetic ganglia. This operation, I believe, will not only save the danger zone of tissue but also cause a marked improvement or a permanent remission in the course of the disease, as well as prevent its progression. One must do the widest possible resection of the sympathetic ganglions removing the inferior cervical and as many dorsal ganglions as can be reached.

My first case was done three years ago. In this patient the condition was confined largely to the right leg and was associated with painful muscle spasm. The patient, a woman, aged 30, could walk only by use of cane or crutch. Diagnostic injection of the right lumbar sympathetics relieved the pain and enabled her to walk without the help of cane or crutch for the first time in months. Lumbar ganglionectomy resulted in complete relief of pain and spasticity, and strangely enough, her arms so improved that she was able to write a much more legible hand. She was also able to knit, which she could not do before. This result on the arms can be explained on the basis of diminution of reflex activity of the entire sympathetic system as suggested by Royle years ago. This patient remains well to date.

Another patient with major involvement in one leg in whom lumbar sympathectomy was performed has obtained very little if any benefit. It would seem that the cervicodorsal operation is the one of choice since in the cases where the upper operation has been done the results have been promising.

Most of my cases have been done within the last eight months and it is therefore too early to report definite results. It can be said, however, that the results have been encouraging. Most of the early cases have shown progressive improvement. Four cases were hopelessly bedridden patients, and in two of these the improvement has been remarkable.

At present my cases are being reviewed and it is hoped that some criteria of selectivity can be found. There can be little doubt that many cases will not be benefited. It is my belief that the occurrence or absence of remissions is of considerable selective value. Where remissions have ceased I incline to question the advisability of operation. To date the poor results have been in those cases advanced to the stage where remissions no longer occur. In one case no remissions had occurred since onset of the symptoms and the entire history covered only about six months from onset to inability to walk. This is a very recent case and it is doubtful that there has been or will be much, if any, improvement. A complete report of all cases to date will be ready for publication soon.

Essential Hypertension

Adson[6], Craig[7], Peet[8], Page and Heuer[9], and Crile[10] all report favorable results in the treatment of essential hypertension by surgery of the visceral nerves. Adson first advocated bilateral intradural section of the anterior spinal roots from the sixth dorsal to the second lumbar inclusive. This results in paralysis of the abdominal muscles and denervation of visceral efferent fibers to the adrenal glands and abdominal viscera. The operation serves a triple purpose: first, to decrease the vascular tension in the large vascular beds in the abdomen; second, by denervation of the adrenal glands to diminish the pressor effects of its secretions; and, third, to decrease intra-abdominal tension by paralysis of the abdominal muscles. It is an operation of considerable magnitude, especially in patients presenting such poor risks as do those afflicted with this disease, and, it must of necessity be accompanied by a rather high mortality rate. Now Adson is attempting to produce the same effects by sectioning the splanchnic nerves and removing the first and second lumbar ganglions. He does this through a high kidney incision with resection of the twelfth rib and a retroperitoneal exposure. He also resects half of the suprarenal gland at the same time. This procedure is divided into two operations one side being done at a time. He believes that results have been equally as good as they were following the former procedure of intradural section of the anterior spinal roots. He feels that the best results are obtained in patients less than forty years of age having a history of short duration and a slow progression of the disease. One can assume that cases in which there is appreciable damage to the heart or kidneys or in which choked disc has appeared are not suitable. There can be no question that surgery should not be attempted in older individuals in whom the hypertension is associated with advanced arteriosclerosis.

Craig's operation differed in that he did not resect a portion of the suprarenal glands.

It is too early to state definitely that anything of permanent value can be accomplished in the treatment of malignant hypertension. The situation here parallels that in multiple sclerosis. In both we are dealing with otherwise hopeless conditions and it would seem that one would be warranted in attempting almost anything that offers a reasonable chance of giving these patients relief. A number of years must pass before we can accurately evaluate results and before cases can be selected in which a reasonably good result can be predicted.

From an anatomic and physiologic standpoint operations of this

type offer more hope than any other type of treatment heretofore attempted.

Pelvic Pain and Dysmenorrhea

Examination of the literature indicates that gynecologists are showing a great deal more interest in the treatment of pelvic pain and dysmenorrhea. Miller[11] has emphasized the fact that a wide resection of the superior hypogastric plexus is necessary. He states that his results have been successful in relieving pain in about 75 per cent of cases. His most satisfactory results have been in cases of inoperable pelvic cancer. Adson[12] feels that these patients should have endocrine studies and treatment. If these fail, diagnostic dilatation and curetment is indicated. When conservative measures fail he advises laparotomy in conjunction with a gynecologist to rule out any lesions which might be responsible for the dysmenorrhea. If no lesions are found, resection of the presacral nerves is carried out.

It would seem that one should have some definite diagnostic measure to determine whether or not the superior hypogastric plexus should be resected. I have described a technic for diagnostic injection of the superior hypogastric plexus.[13] By means of this we are able to select for operation those cases in which we can expect relief following surgical removal of the plexus. In functional dysmenorrhea the superior hypogastric plexus is injected while the pain is at its height. If pain is relieved for the duration of anesthesia of the plexus and then recurs, we may be quite certain that resection of the plexus will result in cure. These patients frequently have one or two periods following the operation during which they have a modified type of pain and then have no further trouble. This residual pain may be due to a conditioned reflex dependent upon the onset of menses. To quote from a previous publication:[13]

> The majority of cases of pelvic pain which do not respond to ordinary gynecological treatments may be relieved by interruption of the afferent pain fibers coursing through the superior hypogastric plexus. Diagnostic injection of the pelvic sympathetic nerves should precede any surgical operation directed toward relief of pain by resection of the superior hypogastric plexus. Cases of functional dysmenorrhea are particularly amenable to this treatment.

My personal interests have been concerned with several other conditions which are amenable to sympathetic surgery, namely, migraine, atypical pains of the face and neck, encephalitis, and the pain of abdominal malignancy.

Migraine

Dandy[14] was the first to report relief of migraine, or hemicrania, by resection of the cervicodorsal sympathetics. Craig[15] has also reported cases. My own experience has been somewhat exasperating.

One must consider the peculiar psychology of these patients. During the migraine attack they are willing to do almost anything for relief, but no matter how much they may suffer during the attack as soon as it is over they become very uncooperative. This makes it difficult to work them out properly and to determine the cause of the migraine. Since many of these patients are allergic I insist upon skin testing, and a careful dietary regime to rule out the factor of food allergy. If this does not suffice the next procedure is the use of gynergin or similar products. If all medical factors have been exhausted and the patient continues to have attacks, diagnostic injection of the sympathetic nerves is advised. Even though diagnostic injection does abort the attack, one cannot be too certain that surgical removal will be effective. At times the injection seems to exert a tremendous psychic effect and operation should be recommended with definite reservations even though the diagnostic injection has succeeded in relieving an attack. Operation should not be recommended without at least the support of a successful diagnostic injection.

I have recommended diagnostic injection at the next attack to several patients: many times these people who invariably have attacks at frequent intervals will remain in the city for several weeks without an attack, and then have one immediately upon returning home. There is no doubt that the migraine attack itself has a tremendous psychic background. For this reason, any surgical procedure directed toward its relief must be somewhat uncertain. A recent patient in whom the attacks were apt to come on either side, but were always confined to one side at a time, was operated following a successful diagnostic injection. The subsequent history is interesting as only one side was done. She has continued to have severe attacks on the unoperated side, but no severe attacks on the operated side. Resection of the unoperated cervicodorsal nerves should make her comparatively comfortable. I believe that if these cases are properly investigated, the majority can be relieved by either medical or surgical means.

Atypical Pains of Head and Neck

Cases of atypical pains of head and neck are interesting and frequently baffling. Some of them can be relieved by sympathetic surgery. The causative factors of this condition are frequently multiple. For example, one recent patient had not only migrainous attacks with pain in the neck and shoulder, but also tic douloureux and associated accessory nasal sinus disease with pain. The nasal sinus disease was treated surgically and several years previously alcohol injection of the second division of the trifacial nerve had

modified the pain. Removal of the cervicodorsal sympathetics in this case promptly relieved all of the pain in the head, neck and shoulder, but left a residual paroxysmal pain in the face typical of tic douloureux which was relieved by alcohol injection of the second division. The patient is now completely relieved of all pain after fifteen years of discomfort. The inferior cervical and first and second dorsal sympathetic ganglions were found to be small, about one-fourth the normal size, and microscopic examination showed marked sclerosis with ganglion cells embedded in scar tissue. This case is particularly noteworthy in view of the fact that there was a definite Horner's syndrome present for many years before the operation.

A second patient gave a history of twenty years of paroxysmal attacks of pain in the right side of the head, neck and shoulder, and a sharp pain in the face associated with a complete Horner's syndrome. Diagnostic injection of the sympathetics during an attack relieved the pain. In both cases a diagnosis of a degenerative lesion of the sympathetic ganglia was made because of the presence of Horner's syndrome. The sympathetic ganglions in the latter case were also found to be small and sclerotic. It is interesting to find that after almost a year of relief this patient has developed a facial pain typical of tic douloureux for which alcohol injection or section of the sensory root has been recommended.

I have not seen similar cases reported in the literature. Both patients had developed a Horner's syndrome during the development of which there was a great deal of pain. In both cases operation revealed small and sclerotic sympathetic ganglions. The pain in these cases is probably due to the degenerative process in the sympathetic ganglions. Why this process should be selective as to location we do not know.

Parkinsonian Syndrome of Encephalitis

These cases have interested me since Royle's report of improvement following sympathectomy. A previous statement that I had never seen a case improved by sympathectomy must now be retracted. About a year ago a case was done utilizing the new anterior approach by means of which it was possible to do a more complete resection of the cervicodorsal sympathetics. The patient was a young woman with a typical Parkinsonian syndrome which rendered her quite helpless because of increased muscular tonus and tremor. I felt that the excessive muscular tonus might be diminished by sympathectomy. Bilateral cervicodorsal sympathectomy resulted in a marked diminution of tonus and also lessened the tremor thus enabling her to do things with her hands that she

had not done for years. While she insists that the tremor is markedly diminished I believe that the benefit is due to the diminution of excessive tonus since the tremor in these cases is the reverse of an intention tremor. She was so pleased with the result that bilateral lumbar sympathectomy was performed with marked improvement in the lower extremities. Another patient, a boy of 19, has also experienced a marked improvement in the ability to use his arms, in lessening of salivation, and an improvement in speech. I have seen other cases in which sympathectomy has been done without improvement. Only cases in which the increased muscle tonus is the major factor should be considered. Another factor is age: the younger the individual the more chance of a beneficial result.

Pain of Abdominal Malignancy

The following case is reported merely to indicate a means of giving these individuals relief by a minor operative procedure. The patient was a man in his late forties with carcinoma of the pancreas upon whom a cholecystogastrostomy had been performed. There was a large tender epigastric mass with constant excruciating pain which required large and repeated doses of morphine. Feeling that the pain was probably transmitted through the celiac plexus I determined to attempt relief by injection of this plexus. Using Labat's technic for splanchnic anesthesia, but going higher, I injected 10 c.c. of 1 per cent procaine into the region of the celiac plexus. Within ten minutes he was completely relieved of pain and in addition I was able to palpate the mass without causing pain. Ten cubic centimeters of 95 per cent alcohol was then injected without untoward results and with complete relief of pain for a period of about six weeks, after which mild pain recurred for a few days. At this time, approximately three months after the injection, he is quite comfortable and requires no medication for pain. This result probably will not be permanent due to the fact that the alcohol was diluted as a result of injection soon after the procaine. A subsequent injection will no doubt be necessary, at which time I will use only alcohol. This case is offered merely to indicate a possible method of giving relief in upper abdominal malignancy by a very simple method.

Technic

The majority of American surgeons use the posterior approach for the removal of the cervicodorsal sympathetic ganglions. I have given up this approach in favor of the anterior approach of Gask[17] which has been previously described.[18] This approach has many advantages over the posterior approach.

For the lumbar sympathetics the transabdominal approach still retains its popularity. However, more and more surgeons are adopting the extraperitoneal approach of Royle which I have modified so that it is now done through a muscle-splitting anterior approach.[19]

It is probable that the recent work on the extraperitoneal approach to the splanchnic nerves and the upper lumbar ganglions will illustrate to others the advantages of this type of approach. I might add that we have been able, in the course of a lumbar sympathectomy for a bowel condition, to extend the exposure over the aorta and remove the descending fibers of the intermesenteric plexus.

SUMMARY

The newer work on surgery of the sympathetic and visceral nerves in cases of multiple sclerosis, essential hypertension and pelvic pain and dysmenorrhea has been discussed.

A few subjects have been discussed in which I have been interested personally in the past year in which surgery of the sympathetic nerves has afforded relief, namely, migraine, atypical pains of head and neck, encephalitis and the pain of upper abdominal maligancy.

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DYSCHONDROPLASIA

A Consideration of Osseous Dystrophies

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AN increasing number of reports of osseous dystrophies affecting the bones of the extremities and the appearance of a few papers attempting to differentiate the large variety of cases of idiopathic disturbance in growth of bone not attributable to any known cause, such as infection, trauma, endocrine imbalance, or vitamine deficiency, are making more members of the profession aware of characteristics of these affections. Several facts, however, indicate that too frequently lack of familiarity with these conditions is the rule. Records of large institutions in which the diagnoses have been made by different staff members show a variety of names for the same affection. Some of the diagnoses are incorrect, showing no knowledge of the pathogenesis or clinical picture. Secondly, there is no generally acceptable classification of diseases characterized by idiopathic disturbances in the growth and development of bones. This is obvious when one even slightly familiar with the different varieties observes the terminology in some of our acceptable nomenclatures of diseases. The criticism must not be attributed entirely to the classified nomenclature, even though there are some rare bone dystrophies that cannot be classified under terms given in the lists, but it is accountable for in part by the fact that our knowledge of these diseases is insufficient. Often there is no distinct line of division between one and another dystrophy.

Whereas certain rather well defined clinical and roentgenologic types are crystallizing in our knowledge, others confusing and less distinct continue to be reported. So little is known about the etiology and pathogenesis that even increments of additional knowledge justify a new discussion. I do not propose to solve these problems, but to discuss one of the more common of these dystrophies through the medium of six cases of a particular type of dyschondroplasia.

Classification. Possibly all the idiopathic dystrophies of long bones can be grouped under the following proposed classification:

1. Dyschondroplasia
   a. Hereditary deforming chondrodysplasia (Ehrenfried)
   b. Dyschondroplasia of Ollier or Ollier's disease
   c. Albers-Schoenberg type (stippling)

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d. Voorhoeve type (longitudinal striation)
2. Achondroplasia
3. Familial osseous dystrophy of Morquio or Morquio's disease
4. Osteogenesis imperfecta—fragilitas ossium—osteopsathyrosis
5. Hypertelorism (with dwarfing)
6. Hyperchondroplasia—arachnodactylia
7. Madelung's deformity
8. Aplasia

Though I do not wish to digress into a detailed discussion of the points of difference and similarity of all the above conditions, brief reasons will be stated for dividing dyschondroplasia into four different types of the same disease. These types are based on roentgenologic characteristics as well as clinical. Ollier[1] gave the name "dyschondroplasia" to a condition characterized by cartilaginous masses in the phalanges and in the metaphyses of long bones, exostoses, and what he regarded as unilaterality of involvement of the long bones, though both hands of one of his patients were affected. The members of the affected side were shorter than those of the unaffected. The clinical condition of multiple exostoses was known even before Ollier, but undoubtedly the importance of Ollier's paper rests in great measure on the x-ray studies, then not long available for clinical use, and his interpretation that beneath the clinically appearing exostoses were lesions, basic and more significant; viz., the incompletely ossified areas in the metaphyses and the enchondromas in the phalanges. His name "dyschondroplasia" is a descriptive one for the underlying pathology and it has been widely accepted. In 1915, Ehrenfried[2] collected 300 articles representing 600 cases of a dystrophy which he called "hereditary deforming chondrodysplasia." He emphasized the hereditary basis and studied in detail in this and later[3] papers the clinical and roentgenologic characteristics. He removed a piece of cartilage from an involved epiphyseal plate to find microscopic evidence of cartilaginous dysplasia. The name used by him, "hereditary deforming chondrodysplasia," is now frequently employed. It is apparent that the usual characteristics of the typical case described by Ehrenfried and the cases described in some detail by Ollier have some points of similarity, some of dissimilarity. Bentzon[4], Ehrenfried, Jansen[5], and Voorhoeve[6] regard them as the same disease, but fewer authors, notably Hunter and Wiles[7] and Cole[8], regard them as entirely different clinical and pathologic entities. In studying pictures and x-rays published by various authors, one gains the impression that true Ollier's disease is unilateral or almost entirely unilateral; heredity is less frequently established, and exostoses are not so prone to occur. But in each condition there is an affection of the epiphyseal line, incompletely ossified metaphyses, and dim-

inished growth. Both are a dyschondroplasia and whereas they seem to present different clinical pictures, mainly because of asymmetry in one and more prominent exostoses in the other, the pathogenesis is indeed similar, there being truly a difference only in degree and incidence of the occurrence of the different pathologic manifestations.

Albers-Schoenberg[9], in 1915, reported an affection of bones characterized by rounded or irregular areas of increased density, affecting mainly the metaphyses and epiphyses, but rarely the diaphyses. Irregularity of calcification demonstrable roentgenologically is one of the characteristics of dyschondroplasia[3, 7] of the hereditary deforming chondrodysplasia (Ehrenfried) type, but it is not as marked in this condition as in that described by Albers-Schoenberg, and other phenomena are more prominent in the former.

Voorhoeve reported the cases of brother and sister who had marked longitudinal striation of the metaphyses of the long bones. The x-rays show unmistakable striation, broad dense lines. The metaphyses were wide and there were small exostoses. But the predominant findings were broad stripes running in the long axis of the bone and representing uneven areas of calcification. He considered the bones of the father of these two patients abnormal, but these were not as evident as in the children. The findings in the father were wide metaphyses and irregular, poorly calcified areas. He regards both the condition he described and disseminated condensation of ossification (Albers-Schoenberg) as manifestations of dyschondroplasia.

In reality all these conditions have characteristics in common. They affect mainly the metaphyses in the region of the fastest growing growth centers of the osseous system; they are characterized by deficiency of ossification and tendency to form osteochondromas, and in many instances heredity may be demonstrated as the basis for their occurrence. Until an etiologic agent can be discovered definitely separating them as distinct entities they may be regarded as dyschondroplasia or disturbances in cartilaginous growth and ossification. Dyschondro-osteoplasia would be perhaps a little more descriptive, because it is not only a disturbance in cartilaginous growth but also in bone formation that is the true basis for the bizarre forms it presents. However, there are enough terms and Ollier's seems to be adequate if those interested in the details indicate the special type.

Multiple exostoses is another closely related phenomena that so far has not been definitely classified in relation to the above disease. Among the six cases studied by me, there was one in a young woman who had an x-ray of the knees because of pain fol-

lowing an injury. Multiple small exostoses were thus accidentally discovered affecting the adjacent part of the metaphyses. Growth had been normal and they had nothing to do with the pain which resulted from a lacerated internal semilunar cartilage with effusion into the joints. She was 34 years of age and ossification was uniform and complete. There was no family history of a similar affection, but the metaphyses in these areas were broad for an abnormal distance up the shaft. X-rays of the wrist were negative, except possibly a slightly longer radius on one side, a finding which by itself would indicate nothing. The broad metaphyses and exostoses are so characteristic of the hereditary deforming chondrodysplasia (Ehrenfried) type of dyschondroplasia that we cannot ignore them or even think so little of these manifestations that the conditions can be entirely separated. Had the condition been a little more marked in this case we would undoubtedly have had a manifest case of dyschondroplasia of the Ehrenfried type. If the case had been studied when the patient was 14 years of age, she probably would have shown more manifest characteristics of dyschondroplasia; viz., poor ossification, because the tendency in the disease is toward more complete ossification later in life. It is doubtful if any cases of multiple exostoses can be thrown out of the category of dyschondroplasia. Frequently heredity can be established, and the exostoses affect generally the metaphyses in the region of the epiphyseal plate where fastest growth is occurring. Unless it can be shown more decisively than has been demonstrated in the past that these conditions are distinct entities, we are safe in assuming that they are the same with one or another or all of the characteristics either accentuated or unexpressed, resulting in the several clinical and roentgenologic types. Multiple exostoses is a diagnosis frequently used for typical cases of dyschondroplasia. In the less typical case, like the above one must be familiar with the characteristics of these diseases to see manifestations of dyschondroplasia, like the telltale broad metaphyses.

There is little difficulty in differentiating achondroplasia and dyschondroplasia clinically or roentgenologically. In the former the short extremities, early union of epiphysis with shaft, arrested hydrocephalus, and trident hand with ossification even in advance of growth in contradistinction to the improperly ossified areas of the metaphysis in dyschondroplasia leaves little in common but that they are both dystrophies, one almost the antithesis of the other.

Hypertelorism, whose main characteristic is abnormal broadness between the eyes, is sometimes characterized by an associated shortness of stature. There is no deficiency of ossification, but rather a

lack of proper longitudinal growth of the bones of the extremities, simulating to some extent achondroplasia.

Morquio's disease [10, 11, 12] is a dystrophy which differs markedly from dyschrondroplasia. It is extremely rare. The patients are weak, kyphotic, short, and poorly developed. The centers of ossification in the epiphyses and carpal and tarsal bones appear late and develop slowly. Growth is retarded. Ossification is uniform, but there is deficiency of calcification affecting not only the metaphyses but also the diaphyses and epiphyses. There are no exostoses and the peculiar appearance of the knock-kneed stance, short trunk, and extremities with feeble musculature permits one who is familiar with its characteristics to suspect it at a glance.

Osteogenesis imperfecta is probably a categorical term including a number of conditions in which there are no abnormalities of form, but deficient calcification of bones. Apparently ossification takes place but insufficient calcium is present uniformly throughout the bones. They are brittle and multiple fractures may be present at birth (fragilitas ossium). The condition may manifest itself later in childhood when insufficient trauma results in fracture.

Hyperchondrodysplasia[13], abnormal idiopathic growth in length of bones, not pituitary gigantism, is extremely rare. It may affect the digits or long bones and is known as "spider fingers"; arachnodactylia.

Madelung's deformity, characterized by bowing at the lower end of the radius, displacing the hand forward, the ulna backward, is uncommon. It is usually bilateral, affecting the distal epiphysis of the radius. The rest of the skeleton develops normally.

Rare indeed is the idiopathic dystrophy of bones of the extremities that cannot be included in the above types. Freund[14] reported a case of unilateral dystrophy of bones of extremities with asymmetry of the face. X-rays show a uniform osteoporosis of the shaft as well as the juxta-epiphyseal areas. Biopsy showed osteitis fibrosa. This may prove to be another type of dyschondroplasia.

Hereditary Deforming Chondrodysplasia

Incidence. In 9,446 admissions to the Hutchinson Memorial Clinic of the Tulane University School of Medicine, there are 9 cases of osseous dystrophies affecting the long bones; hereditary deforming chondrodysplasia, 5; achondroplasia, 3; hypertelorism with short stature, 1; true Ollier's disease, none; Morquio's disease (familial osseous dystrophy), none; osteogenesis imperfecta, none. There are other cases of disturbances in growth of long bones,

but none in which the cause was unexplained; i. e., they could be placed in the etiologic category of trauma, infection, deficiency diseases, or endocrine deficiency (cretinism). This incidence tends to show that if we are correct in our diagnoses of hereditary deforming chondrodysplasia it is one of the most common of the idiopathic developmental defects of the long bones of the extremities. The author has had under his care one other case elsewhere.

Clinical Characteristics. Studies of distribution between sexes show that males are more commonly affected. The proportion of males to females in different series varies from 5 to 2 to 3 to 1 [15, 16]. A history of other members of the family being affected can be established very often. Ehrenfried in a study of 236 cases from the literature found that heredity could be demonstrated in 176 (74.6 per cent). It was not found in 18 cases, and in the remaining 42 heredity was uncertain or not mentioned. According to Hunter and Wiles[7], dyschondroplasia of the true hereditary deforming dyschondroplasia type may be unilateral, but practically all cases are bilateral.

In six cases studied personally the earliest manifestation of the disease recorded for one of the patients was at 2½ years. Usually the irregularities on the epiphyses are noticed between the first and fifth year, and in one case described by Ehrenfried[8] deformity was first noticed when the patient was nine months of age. In one of our patients it was not noticed before 5 years of age. The first signs appear some time after birth and are nodular enlargements in the region of the metaphyses, usually most conspicuous on the metaphyses of long bones in which growth is most rapid. Asymmetry may be an early symptom in unilateral cases. Osteochondromas usually increase slowly in size up to the time of cessation of growth of the skeleton. They remain stationary after that and resumption of growth in adults is suggestive of malignant change. Some of the osteochondromas grow faster than others and occasionally one reaches a large size. An osteochondroma removed from the femur of one of our patients measured 8 by 8 cm. Growth may be arrested at any time and cases have been recorded in which an osteochondroma diminished in size. Those lesions are firm and painless, but may interfere with joint function, especially rotation of the forearm. Paralysis from pressure of an exostosis on a nerve may occur and in some instances aneurysms have developed. The mass may be so large as to interfere seriously with such fundamental function as walking, a complication present in one of our cases. According to Ehrenfried, malignant change or rapid growth occurs in 5 per cent of cases, but he does not say that actual malignant degeneration occurs in 5 per cent: some grow rapidly without being

malignant. One gains the impression from reviewing the literature that malignancy is not a common complication.

As the affected individual becomes older shortness of stature is noticed. According to Ehrenfried, this is the rule and in the family history of one of our cases the affected individuals were invariably short (about 5 feet, 1 inch), whereas the unaffected relatives were invariably taller. In Ollier's type of unilateral dsychondroplasia the affected limbs are shorter than the unaffected. Various deformities may be noticed besides the exostoses. Among the more common are knock-knees, long metaphyses; i. e., the broadness of the metaphyses extending an abnormal distance upon the diaphyses; valgus deformity of the foot, because of short fibulas and curving tibias, throwing the joint surface out; bowing of radius and short ulna

Fig. 1. Patient, 36 years of age, with dyschondroplasia (hereditary deforming chondrodysplasia type). His son had the disease, no other known case in the family. There is marked varus deformity of elbows and incomplete adduction of the right shoulder, due to the osteochondromas at the upper end of the humerus.

with ulnar deviation of the hand, and dislocation of the head of the radius, which according to Keith[17] occurs in one third of the cases.

Bilateral involvement is the usual finding in dyschondroplasia, but among the cases first reported by Ollier, one was entirely unilateral, and one was unilateral except that the opposite hand was affected. The true unilateral type is the Ollier type of dyschondroplasia.

These patients have normal mentality and there is no evidence of arrested internal hydrocephalus such as occurs in achondroplasia. The face and skull usually show no clinical evidence of the disease,

but asymmetry of the head is sometimes found, and in one of our cases the cartilaginous septum of the nose was abnormally broad. Osteomas of the bones of the head occur but rarely[15]. The patients' musculature is good. They are well developed, but do not seem to have the athletic tendency frequently exhibited by achondroplastic dwarfs. They live a normal, unrestricted life, apparently unashamed of their deformities; but they are puzzled by them and are normally anxious for their offspring not to be so affected.

Pathology and Pathogenesis. Hereditary deforming chondrodysplasia affects the long bones of the extremities in the juxta-epiphyseal areas. Osteocartilaginous outgrowths occur and the sites of predilection for these are the lower end of the femur, the upper end of the tibias, the lower end of the radius and ulna, and the upper end of the humerus; in other words, near the epiphyses at which the most rapid growth is occurring. The skull, spine and epiphyses of the long bones, and the carpal and tarsal bone are infrequently affected. But in one case studied by the author there is uneven calcification (falling snow appearance) in the calvarium. This appearance is not marked and it is the only one found recorded as being thus affected so I do not consider it a characteristic of the disease. The epiphyses themselves in some of the cases studied by me showed striping or irregular calcification. Their centers of ossification seem to appear properly and they unite early with the shaft. The metaphysis or juxta-epiphyseal portion of the shaft is as broad as the epiphysis and this broadness does not rapidly fade into a normal shaft, but the expansion is maintained for some distance up the shaft. The middle of the shaft seems to be normally ossified and have normal relationship between cortex and medulla, but in the wide juxta-epiphyseal portion the cortex is extremely thin.

The marrow cavity at the ends of the long bones is not well formed but is packed with the same unossified cartilaginous and fatty tissue that constitutes the central part of the osteochondromas. Grossly the tissue is soft, almost gelatinoid but gritty, and on cut surface is moist, fatty, and contains numerous small spicules of osseous tissue. In studying an osteocartilaginous tumor removed at operation in one of our cases, cartilage was found on the surface but not in the central areas.

The superficial part of the excrescences is frequently a layer of cartilage, though some have a definite, thin, bony cortex. In the central portion is a soft cartilaginous and fatty material in which are fine spicules of bone. These ecchondromas or osteochondromas have several other interesting features. First, though they may not project out further than the projected edge of the epiphysis they frequently do, an important fact in discussing one of the theories

that have been advanced for their formation. Secondly, their apex is usually pointed toward the central portion of the shaft, and thirdly, they are frequently the point of muscular attachment; for example, some of the largest exostoses appear at the lower mesial aspect of the femurs, less marked on the outer side. I have carefully studied exostoses to see if in any instance they pointed toward the epiphyseal line and if they invariably pointed in the direction of the force of muscle action. In the head of the fibula if exostoses pointed in the direction of muscle pull, they should point toward the biceps and thus toward the adjacent epiphyseal line. In one instance there was a suggestion of a small exostosis pointing upward in the head

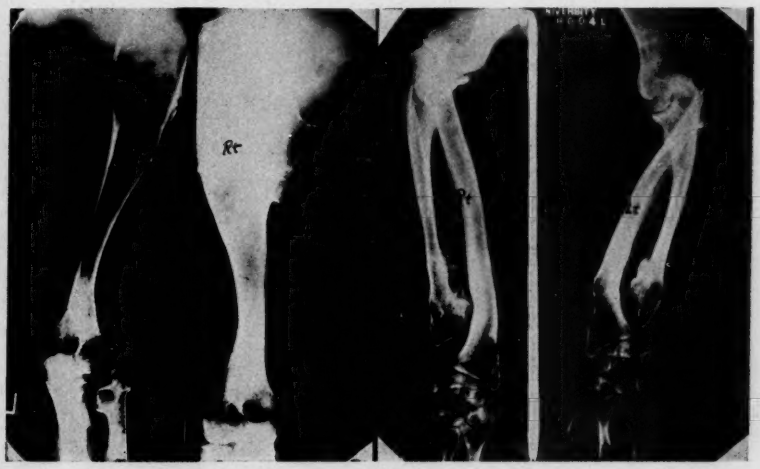

Fig. 2. X-rays of arms showing broadness of metaphyses extending abnormally high on the diaphyses, exostoses, dislocation of the head of the radius, arrow head deformity of the ulna, separation of radius and ulna.

of the fibula. This was not definite and in all other instances the apex was directed away from the adjacent epiphyseal line or straight out from the shaft, not toward the epiphysis. What the exact significance of this is, is not known, but it is best explained by Jansen's theory[5] of imperfect tubulation (v.i.).

The phalanges show enchondromas, or incompletely ossified areas. Authors who describe the lesions of the phalanges (Ollier[1], Ehrenfried[2, 3], and others) frequently give the impression that these changes in the phalanges are distinct from those occurring in the long bones of the extremities. To me such a differentiation does not seem necessary, for x-ray appearances and consideration of the anatomy of the development of the bones show the incompletely

ossified areas to be close to the epiphyseal lines and these areas are in miniature the same changes occurring in the juxta-epiphyseal areas of the longer bones. The phalanges are rarely expanded by the enchondromas. Small exostoses may be present, as in one of our cases. The exostoses may be at the distal or proximal ends of the phalanx, more marked at the metaphysis. The incompletely ossified areas generally occupy the proximal juxta-epiphyseal portion of the shaft of the phalanges and metacarpals of the thumb and the distal part of the other metacarpalis. This is anatomically interesting because the growth line of these bones is single, proximal in all the phalanges, and in the metacarpals of the thumb and great toe, distal in the other metacarpals and metatarsals. Exostoses are less apt to occur on the phalanges than the long bones. Here the growth is relatively slower and such marked developmental defects are not to be found since, as has been pointed out, the greater abnormalities are more prone to occur at the site of the fastest growth in length of bone.

Characteristic changes frequently, not invariably, occur in the long bones of the forearm. The distal epiphysis of the ulna may be poorly formed or even completely absent. Growth in the ulna frequently is not proportionate to that of the radius and the former is relatively short. I would call attention to the fact that the distal radio-ulnar articulation frequently is separated. The growth of exostosis in the interosseous space or the marked relative shortening of the ulna with bowing of the radius may be responsible for springing of the two bones, but the interosseous space between tibia and fibula may be wider than normal when the abnormality cannot be explained on overgrowth of one bone, but on the presence of excessive osseous tissue or osteochondroma between them apparently pushing them apart. In some instances a rough area appears on the ulnar aspect of the radius near the lower end, but above the true joint. This apparently is an attempt at the formation of a new joint. The head of the radius may be entirely dislocated. Marked varus deformity of the elbow may result from disproportionate growth of the radius and ulna.

The short stature in these cases might at first sight be considered a family trait, but this alone is not the cause of shortness as evidenced by at least two facts; namely, that in the family of a case studied by me, the members of the family affected by the disease were short, while those who did not show evidences of it were invariably taller. Another fact which is of even more importance is that in dyschondroplasia of Ollier's type, the involved side is shorter than the uninvolved. This shortening is due to lack of growth and to a premature union of epiphysis and diaphysis.

Pathogenesis and Etiology. Various theories have been advanced to explain the etiology and nature of the disturbance resulting in the various clinical characteristics. Some of them deserve consideration, others having little soundness scarcely deserve mention. According to Arthur Keith[17], John Hunter realized that in the growth of bones, increases in length were accompanied by a secondary change, remodelling and reshaping of the new bone and incorporation of it into the shaft. Keith elaborated on the fundamental hypothesis and believed that the disease which he termed "diaphyseal aclasis" (hereditary deforming chondrodysplasia) was occasioned by a failure of the remodelling process (the formation of the small diameter shaft from the broader epiphysis as the bone grows),

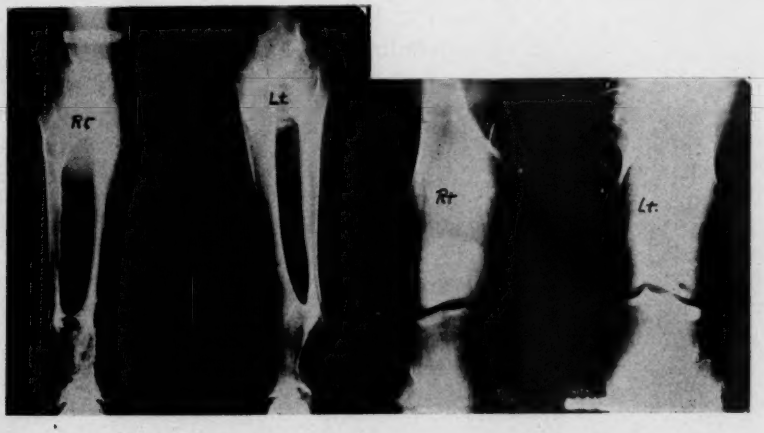

Fig. 3. X-rays of knees show the broad metaphyses, incomplete ossification, and exostoses. The dotted lines indicate the potential breadth if no tubulation had occurred, and show how partial failure of absorption explains to some extent the occurrence of exostosis. On the other hand, a portion of the exostosis extends beyond the projection limits through which the bone developed. Growth in the exostosis itself must explain this additional extension. There is fusion of the upper and lower ends of the tibias and fibulas and deviation of the ankle joints outward 45 degrees from the normal, with a short fibula. Abnormal striations are present. The negatives have been retouched lightly in places to preserve detail.

noting that the bone at the metaphysis and for some distance up the shaft was as wide as the epiphysis. Keith stated that the bone changes seem to be confined to places where the formation of bone within the cartilage later comes to be covered with periosteum. He believed that the periosteal covering of the metaphysis ceases to keep up with the growth of the epiphyseal cartilage, thus leaving an uncovered cartilaginous growth which has no periosteal covering,

which he considered the agent in the remodelling process. An objection to this theory is the fact that areas more distant from the epiphyseal line than other areas in which the ossification had progressed more normally are covered by periosteum and it could not be due to uncovered cartilage that failure of ossification or remodelling occurs unless we hypothesize that certain sporadic areas are deficient, and not merely the terminal portion of the metaphyseal covering.

The latter explanation only initiates more objections; one, that the line of demarcation at the edge of compensatory function from surrounding zones adequately covered by functioning periosteum is not evidenced by patterns which could be expected by supposing a sporadic deficiency in periosteum. Keith failed to take into consideration that in these individuals there is frequently short stature and he did not stress the fact that in this condition called by him "diaphyseal aclasis," ossification, an earlier process than remodelling, is delayed and imperfect and that if the two processes are to be dissociated that the failure of remodelling may be consequent on a failure of ossification, or that both processes may be deficient. Jansen, in a more elaborate development of the theory, thinking clearly in terms of dissociation of function, names six different processes which must occur harmoniously in order for bone to develop normally. He thinks that retardation of differentiation of cells leads to cartilaginous metaphysis and that partial retardation of tubulation results in exostoses. Tubulation is the remodelling process by which the broad juxta-epiphyseal portion of the shaft after a period of time in which growth occurs becomes the more narrow shaft. Jansen's theory is interesting and explains very well the pathogenesis of the condition if we can or must for simplicity's sake differentiate processes in the growth of bone. In reality they probably cannot be differentiated absolutely, but it helps to think of them separately and to explain a clinical state on the basis of one or more of these processes failing partially or completely. Attention has been called repeatedly to the fact that exostoses occur in the disease at the ends of the shaft of the bones undergoing the most growth. That growth of so-called exostoses ceases when maturity is reached. It has also been observed by Jansen that tendinous insertions may have initiated exostoses and as the bone grew the tendon became separated from the mass which may or may not have increased in size. Exostoses do not always point in the direction of muscle pull and bone does not grow according to least resistance.

Bentzon[4] attempted to explain dyschondroplasia on a disturbance of the sympathetic system resulting in a lack of vasoconstrictor

action on nutrient vessels to bone. In the first place, when other evidence of disturbed sympathetic function besides disturbances in bone growth are lacking, we must presuppose that the disturbance of the sympathetic function must affect no other vessels but those entering the nutrient foramen, which, in a widely disseminated skeletal disease, would have not one central, but various peripheral, selected foci of action; and secondly, our present knowledge of the effect of hyperemia or ischemia on growing bone does not give us evidence satisfactory for explaining the various lesions found in the bones in this disease. To obviate this necessary supposition, Bentzon presumed, without evidence to support it, that the sympathetic supply to the vessels nourishing the bone had a separate central division from that of the nerves supplying the vessels to the soft parts. His experimental work is unconvincing, but is frequently quoted, more frequently perhaps than it deserves. I agree with Richard, Dupuis, Roederer and Froyez[18] that Bentzon has failed to produce a condition analogous to dyschondroplasia. As a matter of fact, Bentzon himself does not claim to have given irrefutable proof of his assumption that a deficiency of sympathetic vasoconstrictor action is accountable for the disease. Without citing his experiments in more detail and leaving the reference for those more interested, let us pass the theory over with the belief that the main reason for giving space to even its limited discussion is the fact that the work has been so frequently quoted.

In his excellent paper on dissociation of bone growth, Jansen[5] declines to accept Bentzon's theory of vasodilatation on the physiologic basis that vasodilatation does not produce effects comparable to the roentgenologic findings and advances a theory of vasoconstriction originating in a disturbance in the central sympathetic system. In the light of physiology and anatomy (unilaterality or contralateral involvement, arm on one side, leg on another), I would reject this theory too as untenable.

Thyroid dysfunction is a theory which has as a basis the skeletal dwarfs of cretinism, but there is none of the other changes which we would expect with thyroid disturbances occurring in the disease. Moreover, the thyroid seems to be normal in size and not hypofunctioning. Endocrine dysfunction would work systemically and dysfunction would not explain unilaterality. Though this theory of thyroid dysfunction is frequently mentioned, we do not see recorded cases that have improved on thyroid therapy, which we certainly would find if those who mention it had enough faith in the acceptability of the thyroid as an etiologic agent to employ measures to correct its dysfunction. In attempting to explain the etiology of the condition we are so far forced to revert again to an unknown

something in the germ plasm, a characteristic predetermined deficiency, inherent in the cell that imparts imperfect development. Conditions termed congenital anomalies arise elsewhere in the body. Unquestionably, dyschondroplasia in many instances is inherited. In a case studied by me, there were 17 out of 62 persons in two generations of one family affected by the condition. Ehrenfried found that 61 to 75 per cent of cases gave positive evidence of inheritance. I have seen two abortive cases, one in a boy in which there was no family history and no clinical sign of the disease, the typical deformity at the lower end of the ulna being discovered in the course of an x-ray examination for fracture. This was the only finding indicative of the disease, but it was unmistakable and was undoubtedly a clinically latent form of the disease. Should that patient's offspring be manifestly affected they probably would have been or still may be considered sporadic cases. The disease may skip a generation, and thus a relevant history of it may be hidden from the living members of the family. The theory that repeated fractures in several generations may impress finally germinal cells, leaving them with an acquired transmissible abnormality, was advanced by Hale[15]. Such a theory needs much proof for its acceptance.

Treatment. None is satisfactory for arresting the disease. Fractures are not common and the patient's activities should be unrestricted. Secord, Wilder and Henderson[19] have found that the administration of thymus extract seems to increase calcification in osteogenesis imperfecta. Possibly it will also help in dyschondroplasia. Osteocartilaginous growths interfering with function should be removed. Possibly this indication will arise for cosmetic reasons alone, or because of size or interference with function. Sometimes relatively small masses should be removed, because they impede joint function, notably in the upper extremity. When the lesions initiate such complications as aneurysm or nerve paralysis, they obviously should be excised. In removal, care should be exercised to do an extracapsular dissection, because if parts of cartilaginous tissue are left recurrence may be encouraged.

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AN INSTRUMENT FOR THE TREATMENT OF TRIGEMINAL NEURALGIA BY ELECTROCOAGULATION

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THE surgical methods in general use for the treatment of trigeminal neuralgia are efficient and the mortality is less than 2 per cent. Unfortunately, many individuals refuse this type of treatment. Chemical injury to the nerve fibers by the injection of alcohol or phenol produces only a temporary block with relief from pain of from approximately three to twenty-four months. The injection of

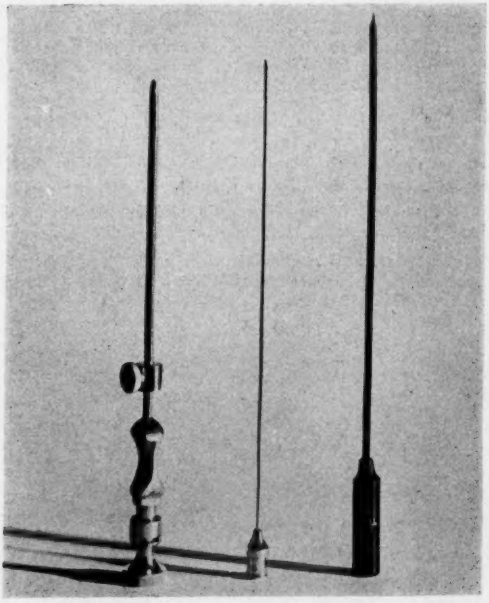

Fig. 1. Needle with stylet, needle for novocain injection electrocoagulating unit.

alcohol in or about the division of the trigeminal nerve may be followed by undesirable or even dangerous sequelae.

An instrument has been designed for permanent destruction by electrocoagulation of the nerve fibers in the maxillary and mandibular branches of the trigeminal nerve. The instrument consists of

Reported before the Medical Association of Florida at sea on April 28, 1936.

three parts. The first part is made up of a needle and a pointed stylet which as a unit measure 14 cm. in length. The needle is composed of a barrel 12 cm. in length, which has at one end a luer lock. Just above the luer lock is a solid concave grooved finger plate. The barrel distal to the finger plate is 9 cm. in length and 2 mm. in diameter. The tip is ground smooth. On the barrel, which is graduated from the tip in millimeters, is a movable guard screw so that the depth of the barrel in the tissue can be accurately

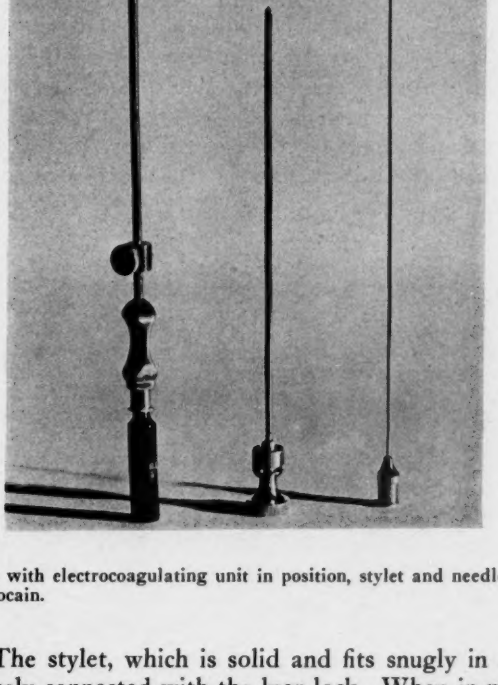

FIG. 2. Needle with electrocoagulating unit in position, stylet and needle for injection of novocain.

observed. The stylet, which is solid and fits snugly in the barrel, can be securely connected with the luer lock. When in position the stylet projects 2 mm. beyond the tip of the barrel. (Fig. 1)

The second part consists of a No. 23 gage needle, 15 mm. in length. The proximal two centimeters of this needle is graduated in millimeters so the distance the needle is introduced beyond the barrel is definitely known. The third part is composed of an electrocoagulating element which is adequately insulated by bakelite. The electrocoagulating unit has a spearlike non-insulated point which protrudes 4 cm. beyond the tip of the barrel. At the proximal part

is an attachment for connection with the electrocoagulating machine. (Fig. 2)

The instrument is introduced with sterile technic under evipal intravenous anesthesia by the usual route for alcohol block of the maxillary or mandibular branches of the trigeminal nerve. When the nerve is reached the stylet is withdrawn and a roentgen examination made for exact localization of the point of the barrel. The patient is allowed to regain consciousness and the long needle is passed through the barrel and 1.5 c.c. of 1 per cent novocain injected directly into the nerve. The degree and distribution of the anesthesia over the surface of the face is then tested by pricking the skin with a sharp pin. When the position of the nerve in relation to the tip of the barrel is thus accurately known, the electrocoagulation unit is introduced directly into the nerve. A minimum current is then passed to coagulate the nerve. It may be that electrocoagulation by this instrument can be substituted for therapeutic alcohol injection in other procedures, as, for example, the interruption of the dorsal sympathetic fibers for the treatment of angina pectoris.

Experimental and clinical observations are being made and will be reported when completed.

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TECHNIC FOR THE INJECTION TREATMENT OF HERNIA

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THE principle involved in the injection treatment for hernia may be said to be the same as that of the operative treatment, namely, that of producing scar tissue at the site of the hernial defect whereby this orifice will be closed and the abdominal contents thereby prevented from passing out of their normal confines.

In accordance with the operative method the hernial sac is first removed. After this is accomplished the floor of the inguinal canal is reinforced by suturing the conjoined tendon to Poupart's ligament posterior to the cord. The internal ring is then made narrower with the aid of a Coley stitch. As the fascia of the external oblique is sutured back into place over the cord, the inguinal canal is closed and also somewhat constricted. A few additional sutures are then placed in the external ring to narrow down this structure. These sutures are usually of catgut and serve to hold these structures in their new position for only a few days, until scar tissue is produced. Thereafter, the newly formed scar tissue keeps the previous defect closed and prevents the recurrence of the hernia.

The injection treatment accomplishes essentially the same objective when properly carried out.

That the injection of a mildly irritating solution can produce scar tissue has been demonstrated beyond a doubt by histologic sections, taken at varying intervals after the injections.[1] The irritating solution is so deposited that the scar tissue which is produced will strengthen the floor of the inguinal canal, narrow the internal ring, constrict the inguinal canal and close down the external ring. When this method is compared with the surgical procedure the only thing which differs is that the actual removal of the hernial sac is not accomplished. There is, however, convincing evidence to prove that the hernial sac is completely closed as a result of the adjacent inflammation.

Thus if it is possible to fulfill these requisites it should be possible to promote a cure of the hernia by the injection method as well as by the operative method.

Solutions injected into the inguinal region will diffuse between the planes of the fascia. This has been demonstrated by the injection

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of a dilute solution of methylene blue into the inguinal region immediately preceding operation. A solution when so injected infiltrates around the cord and passes around the internal ring. The greatest concentration of this solution lies within 1 or 2 cm. from the site of its deposition.

These facts have guided us in developing the technic which has been used successfully at the hernia clinic of the Minneapolis General Hospital.

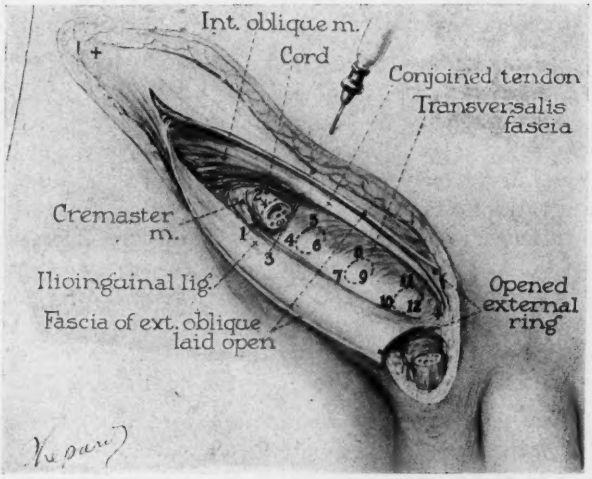

Fig. 1. Indirect inguinal hernia.
Note the obliquity of the needle as it passes through the skin and fascia to approach the under surface of the cord at the internal ring. Injections are made circumferentially around the cord so as to constrict the internal ring and inguinal canal as indicated by numerals 1 to 12.

Technical Precaution

After having been previously fitted with a truss and having worn it for a week or ten days, in order to become accustomed to it, the patient is then prepared for the injection.

He is placed supine upon the table and the hernia completely reduced. If the hernia cannot be reduced, or is reduced with difficulty, the foot of the table may be elevated to facilitate reduction. Complete reduction must be definitely confirmed and if this is impossible the injections must not be started.

The skin is sterilized with an antiseptic solution.

As the injection is made the needle (2 or 2½ inch, 22 gage) is directed caudalward. Therefore in order to enter the inguinal

canal the needle must enter the skin a few centimeters cephalad to the point at which it passes through the fascia. This detail in technic is of utmost importance in avoiding an intraperitoneal injection. If the needle were inserted perpendicularly to the skin, or from below upward, it is conceivable that it might pass too far and enter the peritoneal cavity, whereas with the needle directed caudalward forming an acute angle with the skin of the abdomen, this danger is eliminated. The needle so directed, no matter how long, would pass anterior to the reflection of the peritoneum.

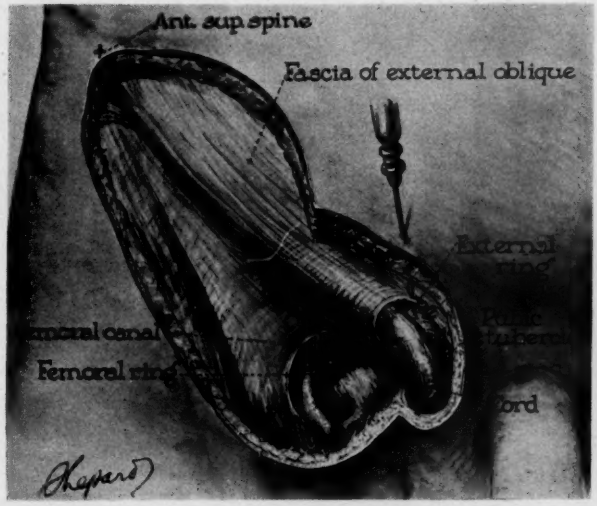

Fig. 2. Method of injecting the external ring.
Note how the needle point passes through the fascia of the external oblique at the margin of the external ring.

When the needle has passed through the skin and subcutaneous tissue its point can be elevated and palpated through the skin. As it passes through the fascia a distinct lack-of-resistance sensation is elicited. If the point is then elevated it cannot be palpated through the skin. This simple test confirms the position of the needle point under the fascia of the external oblique. It is in this location where it is desired to be placed.

The index finger of the left hand is kept constantly in the external ring, through the invaginated scrotum, while the injection is made with the right hand. This procedure also aids in determining the position of the needle point, for as in past pointing, unless one has lost their position sense, the position of the needle point can be very accurately estimated.

Indirect Inguinal Hernia

In treating an indirect inguinal hernia it is necessary to close off the internal ring, constrict the inguinal canal and close down the

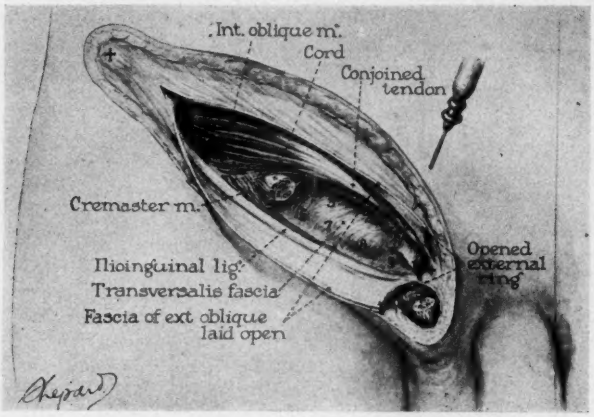

Fig. 3. Direct inguinal hernia.
Note how the needle point passes down to the plane of the transversalis fascia. Numerals 1 to 10 indicate the sites of injection in the floor of the inguinal canal, under the cord, from the conjoined tendon above to Poupart's ligament below.

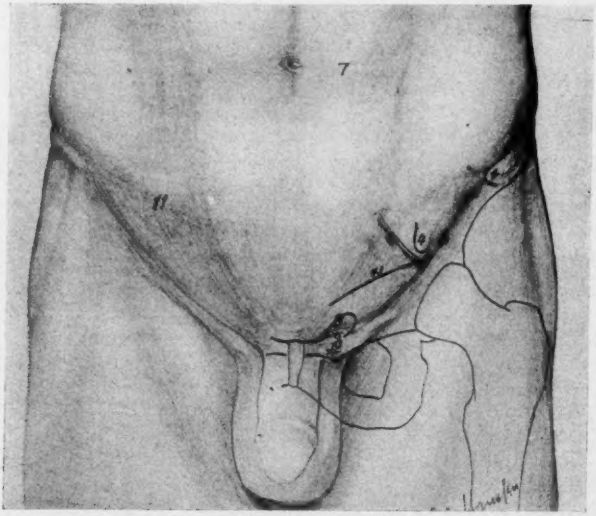

Fig. 4. Topographic landmarks of the inguinal region. 1. Anterior superior spine of the ilium. 2. Pubic tubercle. 3. External inguinal ring. 4. Internal inguinal ring. 5. Ilioinguinal ligament. 6. Inferior epigastric artery. 13. Conjoined tendon. These landmarks must be identified before injections are started.

external ring. This is accomplished by placing the first three injections around the internal ring, in a circumferential manner as indicated by numerals 1, 2 and 3 (Fig. 1). Subsequent injections are placed around the cord in the inguinal canal as indicated by numerals 4 to 12. This procedure serves to constrict the internal ring and the inguinal canal. Those injections placed most superficially, under the fascia of the external oblique, will also serve to obliterate the hernial sac by extension of its proliferative inflammation into the sac. The external ring is made narrower by placing three or four injections at the margin of the ring immediately beneath the fascia of the external oblique muscle as indicated by numerals 11, 12, 13, 14 and 15 (Fig. 2).

It is also well to place a few additional injections deep down in the plane of the transversalis fascia as indicated in Fig. 3 so as to strengthen the floor of the inguinal canal. These injections produce the same effect as is accomplished in the Bassini operation when the conjoined tendon is sutured to Poupart's ligament.

Direct Inguinal Hernia

Direct hernias are treated by placing the injections deep in the plane of the transversalis fascia extending from the conjoined tendon to Poupart's ligament, posterior to the spermatic cord, in the floor of the inguinal canal (Fig. 3). The second layer of scar tissue is developed in the plane of the fascia of the external oblique at the margin of the external ring as indicated in Fig. 2.

Solutions Used

A number of different solutions have been placed on the market which have for their purpose the proliferation of scar tissue, through their irritating effect, when injected into the subcutaneous or fascial tissues. A mild soap solution (sylnasol) has proven to be the most satisfactory in our hands. It produces scar tissue, it is non-toxic and it is comparatively painless. Tannic acid-alcohol solutions (proliferal, Pina Maestra's solution and others) have been tried and found to be effective in producing the desired end result but the pain produced by these solutions has been an objectional feature.

The injections may be made as often as every other day and unless the reaction becomes too severe it has been found preferable to follow this plan in some instances, because patients then do not become so easily discouraged if the procedure should necessitate a large number of treatments.

At the first injection 1 c.c. of sylnasol or proliferal may be given.

Subsequent injections up to 3 c.c. may be used if the patient is found to tolerate them satisfactorily, but if the pain is too great, the dose is decreased. If the more irritating solutions are used such as McDonald's solution, Mayer's solution, etc., the dose should not exceed 6 to 8 minims for each injection. Small hernias may require no more than six or eight injections but the larger ones will require from ten to twelve or more injections before closure is effected.

Sylnasol is the trade-name of sodium psylliate prepared by G. D. Searle and Company.

McDonald's solution contains phenol, alcohol and tincture of thuja in high concentration.

Mayer's solution contains zinc sulfate.

After-care

After the injection has been made the truss is reapplied while the patient is still on the table. It must then be worn day and night until the entire series of injections have been completed. No testings, by having the patient cough, should be made until the series of injections have been finished since any protusion of the hernial contents will tend to redistend the hernial orifice, and defeat the purpose of the treatment.

After the closure appears to be accomplished, and sufficient scar tissue has been produced, the patient is required to wear the truss only during the time that he is up and about. This procedure must be followed for two to three months and at the end of that time the patient is re-examined for any evidence of incomplete closure or weakness at the hernial orifice. If any weakness is present a few additional injections may be given and the truss must then be worn for an additional two or three months after the last injection. At this time it may be gradually removed within the next three weeks.

Abdominal exercises are recommended for the purpose of strengthening the muscles of the lower abdomen. This is very well accomplished by having the patient lie in bed, raise his legs slowly, abduct them from the hips and return them to the original position. By repetition of this exercise, 12 or 15 times twice daily, abdominal musculature is soon satisfactorily strengthened.

Comments

The injection method is most applicable in the direct and indirect inguinal hernias but we have employed it to considerable advantage in the treatment of femoral hernias, small umbilical hernias and small postoperative incisional hernias. However, we have not be-

come as enthusiastic about it in these cases. These details are being dealt with in subsequent papers.

This type of therapy is definitely not to be used in any hernia which cannot be maintained constantly in a reduced position with a suitably fitting truss. Likewise the injection treatment will probably not prove satisfactory in hernias in which the external ring measures more than 3 cm. in diameter. This method is contraindicated in any individual who has ascites, chronic bronchitis or asthma which cannot be controlled, hypertrophy of the prostate gland causing obstructive symptoms and in any condition where there may be undue strain on the abdominal musculature. This type of treatment has proven to be discouraging in very obese individuals because of the uncertainty in maintaining reduction of the hernia. It has been our practice to have these patients reduce their weight before injections are begun.

Conclusions

A careful consideration of the anatomic conformation and superficial landmarks will aid in determining the method of approach and the sites of injection when treating a hernia by the injection method. The minor details of the technic may vary in the hands of different individuals but certain underlying principles and precautions which have been noted above should be constantly observed. The technic which we have described appears to afford the greatest safety, the most satisfactory approach, with the least amount of difficulty, and the best end results.

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THE USE OF THE TIME FACTOR IN PERITONITIS

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IT has long seemed to me that in the maze of articles dealing with the various types of abdominal infections we have become so absorbed in the details of particular conditions considered that we have lost sight of the unchanging fundamental facts. Infection in the abdomen, no matter where located, nor what its source, obeys certain laws. These laws are not different from those governing infection elsewhere. The progress of infection is modified, however, by many things but it should be clearly understood that these are simply modifications of details and not changes in fundamentals.

Some of the modifying factors are, of course, due to anatomic variations, others to differences of virulence or to variations in the resistance of the body to infection. Occasionally, as in perforations of the stomach and duodenum, chemical insults to the peritoneum are such important factors that they dominate and therefore modify the treatment. Similarly in gonnorrheal salpingitis the high resistance of the body completely changes the treatment.

In appendiceal infection there are fewer of these unusual influences and the anatomic arrangement might be termed "standard" for the abdomen. In addition, rarely, if ever, do extraneous factors seriously modify the course of events. For these reasons appendiceal peritonitis will be primarily discussed. However, it must not be forgotten that quite occasionally, as in both the upper abdomen and the pelvis, secondary elements, normally of so little importance, become paramount and dictate the course to be pursued.

In the very young and to a lesser extent in the aged, the progress of appendiceal infection can not be considered "standard." In children the lack of a developed omentum, which is a tremendous aid in controlling abdominal infection, and in the aged the lack of resistance on the part of the tissues profoundly modify the course of a peritonitis. Such modification demands variations in treatment to meet the special conditions existing. However, my experience in the extremes of age has not been nearly so unpleasant as that usually reported in the literature.

Following any contamination of the abdominal cavity, no matter what the source, the peritoneal cavity for many hours remains simply contaminated; in other words there is no peritonitis in the true sense of the word. A reaction in the abdomen has begun and

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the foundation is being laid for the fight against a peritonitis. If the source of the contamination is maintained, or if there is dead tissue to act as suitable food within the peritoneal cavity for the bacteria to live upon and multiply in, peritonitis will inevitably result. If there is no food, or if the source of the contamination does not continue, and providing the dose has not been overwhelming, a true peritonitis will not develop.

With the development of peritonitis there is initiated a twofold defense on the part of the body. First the peritoneum prepares to localize the infection to one or more areas, where the contamination was grossest, and second the body attempts to build an immunity to the specific organism involved. It is extremely important to remember that these two efforts go forward at the same time. They differ in no way from the developments accompanying the formation of a boil and they are hindered by exactly the same factors which hinder the defenses of the body against the infection under the skin.

Practically every one realizes that it is not good to rub a boil. Physicians recognize that this is because mechanical movement of the infected part interferes with the building of nature's wall around the infected area. However, it took the gynecologist or early surgeon many years to learn to wait in cases of acute salpingitis until two things had been accomplished: first the infection had been localized to the pelvic organs, and second the immunity of the body to the infection had been considerably increased. It differs in no way from the old country doctor's statement that he wanted to "wait until the boil was ripe." With the exception of Ochsner and a few others it has taken surgeons until recent years to apply this same knowledge to the ruptured appendix and to other infections in the abdomen.

When the appendix first ruptures a graduated, diffuse contamination of the peritoneal cavity usually occurs. It is of a grossness in some inverse ratio to the distance from the source. If this rupture occurs in so short a time that nature has been unable to initiate the process leading to the formation of adhesions a constant source of infection for the entire cavity is present and a peritonitis which is truly general in type will result. These are the most dangerous of all cases. On the other hand, if due to previous attacks or due to a more slowly developing perforation of the appendix, limiting adhesions are in process of formation, when the appendix ruptures a contamination of the entire cavity may occur but the source of the contamination will soon be choked off. When the cessation of this stream of infecting material is attained by the limiting adhesions, the remainder of the abdominal cavity will destroy the

contaminating organisms and a general peritonitis will be avoided. I wish to emphasize the point that this will occur in the vast majority of cases.

It is obvious, then, that there is a time element in the development of peritonitis since a contamination is not immediately followed by peritonitis. It is probably this factor that has led to more confusion than any other one phase of the question. If one operates upon a ruptured appendix while the peritoneal cavity is still in the early contamination phase the stream of infecting material is stopped by the removal of the appendix. Peritonitis therefore does not develop and, barring accidents, the patient recovers. But if the operation occurs a little later, just after the peritoneal surfaces themselves have succeeded in stopping the contamination and at the height of the effort on the part of the peritoneum to kill out the organisms, the operator inevitably produces a second contamination. This, only too frequently, when added to the first, is sufficient to insure a true peritonitis. I am sure that it is often even worse, completely overwhelming the patient.

If, on the other hand, operation is delayed until the peritoneal cavity has to some extent overcome the infection and simultaneously has localized it in one or more places, a relative immunity will also have been established. Operation at this time can be done more safely. If at operation the clean peritoneal cavity is again soiled it need not be drained because it will recover, just as it did the first time. The infected areas and abscesses can therefore be opened and mopped out and the omentum, or big gut, placed in such a way as to maintain the practical extra-peritonealization of the infected areas which nature has attained. These areas can then be effectively drained. When the omentum is pulled down over the defect in nature's defense wall it can be made to plug the opening very quickly by placing against it a small fluff of gauze on the end of a cigaret drain. It is important to realize that cigaret drains so used are in no sense a drain and that the abscess cavity through which it may run is to be adequately drained with a soft rubber tube. However soft the tube may be it is still a harsh substance and its contact with the intestinal side of the abscess wall should be avoided. This can frequently be done by placing the stem of the cigaret drain between the tube and the gut side.

But there is another factor which may make this an extremely dangerous procedure. If the dead tissue, such as the necrotic appendix is removed, the fluid pus in the abscess mopped out carefully and the cavity left looking clean, there will not be a constant flow of infection through the incompletely restored defects made by the

operator in nature's wall of defense and the peritoneal cavity will remain simply contaminated. Leave, however, blood clot or necrotic appendix itself, and no amount of omental arrangement or drainage has more than an off chance of keeping within bounds the multiplication of bacteria upon the food so offered them. The continued addition of infective material to the free peritoneal cavity through the surgically damaged wall of defense will be insured and a spreading peritonitis will follow.

With this exposition let us turn to its clinical interpretation and then for confirmation to a few statistics. It would seem to follow that there should be a relatively safe period, immediately after rupture of the appendix, during which it would not only be safe to operate, but drainage might even be unnecessary. It seems to us that this period should be very short but recent work indicates a much wider margin than one would suppose existed. For those of us not situated so that experimental clinical work may be done, it would certainly be better not to rely on this factor too strongly for the present. On the other hand, when an appendix is definitely recognized as being ruptured, it should certainly be safer to aid nature's efforts at localization by absolute rest. Auxiliaries to rest at this stage are morphine and the use of any agent which will make the patient more comfortable, such as hot compresses on the abdomen. Avoid anything producing peristalsis. Hence nothing should be given by mouth and continuous suction drainage of the stomach and duodenum should be instituted. If this is followed, in the course of 24 to 48 hours the extreme rigidity over the abdomen will be found to lessen and become localized over the areas where abscesses are in early stages of formation, and the distention will tend to subside. In addition within this period of time some immunity will be attained, the facial expression will become more normal and the pulse will improve, becoming quieter. Occasionally more than 48 hours, perhaps as long as 72 hours, will be required for this change to occur. If waiting is prolonged these patients will then begin an irregular up and down course, responding to leaks in the defensive localizing wall as they develop and there is no occasion for such a state of affairs to be permitted. I cannot emphasize too strongly that the operation should be done at the time when localization has been attained and immunity has been to some extent developed.

On the statistical side we have proof, I believe, of these conclusions. From the recent literature Stanton collected the following table of cases of acute appendicitis, ruptured and unruptured:

TABLE 1

Day of Attack Operated Upon	*Number of Cases*	*Number of Deaths*	*Mortality Percent*
1st	1507	20	1.3
2nd	912	33	3.6
3rd	663	56	8.9
4th	365	46	12.9
5th	442	49	11.6
6th	346	29	8.4
7th, 8th or 9th	178	5	2.8
10th or later	288	7	2.4
Total	4692	245	5.22

You will notice that where operation was done on the third, fourth and fifth days of illness the mortality was high. This is because, in a high percentage of cases, operation occurred before the abdomen had overcome the diffuse contamination and while immunity was still almost non-existent. By the seventh day the majority of patients had accomplished localization and immunity and the mortality dropped. It may be argued that both the number of perforations and the time of rupture in these cases is unknown but I would like to point out that inequalities from this factor would be balanced by similar inequalities from the same factor when any two days of the illness are compared. I have taken this table from one of many similar ones simply because it was well arranged for my purpose. While this group shows a mortality of 5.22 per cent for acute cases only, Finney has recently reported 3913 cases, including chronic and recurrent appendicitis, with a mortality of 2.32 per cent over all and 4.92 per cent on acute and ruptured cases. We cannot regard 5.22 per cent therefore as an unduly high average.

The following tables of perforated appendix cases from my own work are selected from the 209 cases of acute appendicitis cared for in which the mortality over all was 0.95 per cent. The basis of selection was the completeness of the record. A fairly high proportion of records were discarded because of inadequacy on points such as day of illness and so on, but no fatalities were among these. There were no patients lost before operation, either included in the tables or excluded from them.

Of the ruptured group 35 were selected for the following tabular study. Nine case records were eliminated because they contained no accurate statement of the date illness began. All of the nine cases so omitted recovered.

TABLE 2

Acute Appendicitis—Ruptured and Unruptured

Cases of frank acute appendicitis treated since 1924	209
Operated	209
Died	2
Mortality per cent	0.95

Of the group there were:

	Number	*Deaths*	*Mortality*
Ruptured	44	2	4.54 per cent
Gangrenous, not ruptured	5	0	0.0 per cent

TABLE 3

Acute Appendicitis—Ruptured
Clinically not localized on admission
Immediate operation

Case Number	*Day of Illness Admitted*	*Day of Illness Operated Upon*	*Localized? No*	*Yes*	*Died*	*Smoothness of Conval*	*Day Dismissed*	*Complications*
1	3	3	*		0	***	18	None
2	2	2	*		0	**	20	Malaria
3	4	4	*		1	****	3	None
4	2	2	*		0	***	14	Infection of wound
5	3	3	*		0	***	18	Diabetes
6	2	2	*		1	****	13	None
Aver.	2.6	2.6	*		33%	***	17.5	

TABLE 4

Acute Appendicitis—Ruptured
Clinically not localized on admission

Case Number	*Day of Illness Admitted*	*Day of Illness Operated Upon*	*Localized? No*	*Yes*	*Died*	*Smoothness of Conval*	*Day Dismissed*	*Complications*
1	6	7		*	0	*	12	None
2	2	3		*	0	**	12	None
3	2	4		*	0	**	15	None
4	3	5		*	0	*	15	None
5	3	4		*	0	**	10	None
6	5	6		*	0	*	20	Infection of wound
Aver.	3.4	4.7		*	0	**	14	

* Localization? at operation, or smooth convalescence.
*** Stormy convalescence.

TABLE 5

Acute Appendicitis—Ruptured
Clinically localized on admission
Delayed operation

Case Number	Day of Illness Admitted	Day of Illness Operated Upon	Localized? No	Localized? Yes	Died	Smoothness of Conval	Day Dismissed	Complications
1	3	4		*	0	*	17	None
2	4	4		*	0	*	13	None
3	4	5		*	0	*	20	None
4	2	2		*	0	*	11	None
5	6	7		*	0	*	14	None
6	4	4		*	0	*	10	None
7	10	11		*	0	*	15	None
8	7	8		*	0	*	8	None
9	10	12		*	0	***	19	None
10	4	4	*		0	*	8	None
11	4	5		*	0	**	17	None
12	3	4		*	0	*	9	None
13	4	4		*	0	*	12	None
14	3	3		*	0	*	7	None
15	5	6		*	0	*	18	Diabetes
16	8	8		*	0	*	26	Infection of wound
17	2	2		*	0	*	10	None
18	1	1		*	0	*	10	None
19	5	5		*	0	*	10	None
20	2	3		*	0	*	21	None
21	2	2		*	0	*	14	None
22	4	4		*	0	*	17	None
23	21	21		*	0	*	15	None
Aver.	5.1	5.3	1	22	0	*	13.9	

* Localization? at operation, or smooth convalescence.
*** Stormy convalescence.

The fact that no patient died when operated upon after the diffuse contamination had been overcome and localization had occurred is tremendously important. Of the six patients operated upon in violation of the precepts laid down, the operation being done in the presence of a general soiling and a spreading infection and without waiting for localization, two died (Table 3) but only one due to a spreading infection. In the group permitted to localize, either by the patient, his family doctor or myself, I can claim the responsibility in only six cases (Table 4).

In Table 5 several patients were operated upon as emergencies after the appendix had ruptured. In these operation was done because it was thought the appendix was unruptured. They all

recovered but in nearly every instance the rupture had occurred an extremely short time before operation, or the rupture was confined by adhesions from previous attacks. In only one was there a true peritonitis. While these patients recovered they all had a definitely stormier postoperative course than did those who were operated upon after deliberate localization was permitted. There was only one case in which the appendix was left. In this patient I feel confident the appendix had completely disintegrated.

It may be objected that because this series is so small it is of no value in substantiating any conclusions to be drawn. While I feel that this objection is not valid it is fortunately not necessary to rely upon these tables alone. Stanton submits tables showing similar differences in mortality.

TABLE 6

Acute Appendicitis—E. MacD. Stanton

	Number of Cases	*Number of Deaths*	*Mortality Percent*
Frank acute appendicitis	1004	40	3.9
Clinical peritonitis on admission	113	20	17.69
Of the 113 cases there were:			
Immediate operation	31	13	42.0
Delayed operation	82	7	8.5

The following table from Guerry shows the same high mortality when immediate operation is done upon clinically diffuse peritonitis cases as compared with a similar group permitted to establish localization. With perhaps one fourth of his cases I had the privilege of assisting in the operation and caring for them postoperatively.

TABLE 7

Acute Appendicitis—LeGrand Guerry

	Number of Cases	*Number of Deaths*	*Mortality Percent*
Frank acute appendicitis	1666	19	1.14
Of the total group there were:			
Ruptured—localized on admission	511	3	0.59
Ruptured—diffuse peritonitis on admission—immediate operation	94	10	10.64
Ruptured—diffuse peritonitis on admission—deferred operation	137	2	1.46
Ruptured—in extremis—appendix not removed—drainage only	12	3	25.0
Ruptured—Total	754	18	2.38

In conclusion, I wish to point out:

1. That all abdominal infections obey certain fundamental laws.

2. That there is a vast difference between infection and contamination of the peritoneal cavity.

3. That the mortality from operations done after localization and relative immunity have been attained will be much lower than from operations performed before sufficient time has been allowed for this to occur.

4. That the removal of the necrotic appendix is of the utmost importance.

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TUMORS OF THE LARYNX

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New Orleans

TUMORS of the larynx represent such a broad field that a lengthy monograph might easily be written without exhausting the subject. This discussion must be limited principally to the types of tumors of which it has been possible for me to obtain records by motion picture photography. Visualization of intralaryngeal lesions will convey a more lasting impression than a mere pen picture.

It is of common knowledge that accurate approximation of the vocal cords together with synchronous vibration and tension are the three essential requisites for good voice production. Any condition interfering with this triad must result in an altered voice. From this we can readily see that any type of lesion springing from the vocal cords or in very close proximity must result in hoarseness.

Intrinsic carcinomas in their very early stages are confined to one vocal cord. A cure is dependent upon an early diagnosis. Invariably the first symptom manifested is hoarseness. Hoarseness may well be defined as the danger signal of the larynx. Because of the fact that it occurs so frequently as a complication of a common cold, little attention is given it. In any case of hoarseness lasting over ten days or two weeks an examination with the laryngeal mirror should be done for it is only by such a procedure that we may hope to make an early diagnosis in cases of tuberculosis, syphilis, carcinoma and tumors of the larynx. Unfortunately all tumors within the larynx do not give us such an early warning symptom. Tumors and lesions occurring on the arytenoids, aryepiglottic folds and epiglottis do not produce hoarseness until well advanced: their presence is not known until they produce some discomfort.

Benign tumors of the larynx present a number of symptoms, hoarseness being the most prominent. However, aphonia, cough, vocal fatigue, dysphonia and dyspnea may occur depending upon the location and size of the tumor. Probably the most significant and serious of these signs is dyspnea. If severe dyspnea is present in any case, whether benign or malignant, a tracheotomy should

Read before the Seventh Annual Assembly of the Southeastern Surgical Congress, in New Orleans, March 9, 10 and 11, 1936.

Two reels of motion picture films, showing various lesions occurring within the larynx before and after operation were presented.

be done before any attempt is made to establish a definite diagnosis. Dyspnea due to benign lesions of the larynx, is probably most frequently caused by multiple papillomas.

Mirror laryngoscopy is the usual method of choice in establishing a diagnosis in the vast majority of these cases. Mirror laryngoscopy in children is usually unsuccessful because of their poor cooperation: direct laryngoscopy is the most satisfactory method in the examination of these youngsters.

Multiple papillomas is the most frequent benign tumor seen. They occur mostly in young children and occasionally produce severe dyspnea. Multiple papillomas respect no portion of the larynx. I have seen papillomas growing from the tip of the epiglottis to the trachea, well beyond the area of the tracheotomy tube. It is only with increasing fear that I continue semi-annually to remove multiple papillomas now extending to the bifurcation of the trachea in a young man 20 years of age. He first developed papilloma at the age of 3. Papillomas of the larynx is considered a self-limited condition by many. Another of my patients, in spite of numerous surgical removals and appropiate medication, continues to develop these growths 9 years after their first appearance. I can assure you that everything ever suggested has been unsuccessfully tried on these two patients. It will be most interesting to watch their further development. In the due course of time I expect to report these cases in detail. Much remains to be learned about papilloma of the larynx. Frequently a patient will get perfectly well with one or two surgical removals, whereas the same technic may be used on the next few cases with repeated recurrences of the growth. Microscopically the lesions are identical, yet one responds to treatment and the other defies all attempts at eradication.

Suspension laryngoscopy is employed for direct vision and the papillomatous masses are pinched and bitten off by a short Mosher forceps. Every precaution is exerted not to create any new raw surfaces and the tissues are handled as gently as possible. The normal structures of the larynx must not be destroyed but the growth must be removed. It is better to leave the base of the growth rather than destroy healthy tissue. As a rule absolute alcohol is applied to the base of these growths. Fulguration was used in some cases with good results. No agent, however, seems to give uniformly good results. Recently small fractional doses of radiation have been used. In three of my cases excellent results were obtained but failures in several others were disappointing.

Multiple papillomas are most frequently found in children. Only occasionally are they found in adults and then they are considered

as precancerous lesions. Papillomas do not recur with the same rapidity in adults as in children. In adults we most frequently find a single papilloma projecting from the vocal cord composed of a central core of connective tissue and covered with stratified squamous epithelium. The surgical removal of this type of papilloma usually suffices to cure.

Fibromas, as a rule, are diagnosed relatively early as they most frequently occur on the vocal cord and the ensuing hoarseness demands a laryngeal examination. If performed in the early stages mirror laryngoscopy usually reveals a small mass on one of the cords. This type of growth, however, is not confined to the vocal cords but may occur in any portion of the larynx, and if sufficiently large it may interfere with breathing. A small tumor on the free edge of the vocal cord may be removed by direct laryngoscopy with excellent results.

Angioma is a relatively frequent tumor seen in those who abuse their voices. It occurs most frequently on the vocal cord and is usually a small smooth red glistening nodule. It is best removed by direct laryngoscopy, the base being touched with trichloracetic acid on a fine tipped applicator. If care and caution are exercised in the use of the voice, angiomas seldom recur.

Hematoma of the larynx most frequently follows external trauma. Recently two such cases have been seen, both resulting from trauma produced by striking the neck against the steering wheel in automobile accidents. Small hemorrhages into the substance of the cords may also appear from any violent use of the voice. Complete vocal rest, ice packs and iodides to promote absorption are advisable.

Vocal nodules are the result of faulty use of the voice and are found on the anterior third of the vocal cords. It has long been recognized that the continued use of the voice by singers, orators, preachers and school teachers frequently resulted in a gradual progressive hoarseness. The onset of this type of hoarseness is very gradual. It occurs most frequently in those who strain their voices attempting to speak or sing in unnatural tones. I have observed vocal nodules in very young children resulting from excessive yelling. Pathologically, vocal nodules represent a fibrosis of epithelial cells on the contact surface of the vocal cords. These prevent the accurate approximation of the cords depending upon the size of the nodules. Most vocal nodules, if seen early and appropriate vocal rest instituted, subside in time. Those which have existed for a long time as a rule do not yield to rest and must be submitted to surgical procedures. The nodule is grasped with fixa-

tion forceps and shaven off with a very sharp knife. The vocal cord must be guarded against trauma.

Endothelioma of the larynx, a rather rare condition, was described by Lynch who reported seven cases. This type of tumor always occurs on the posterior portion of the larynx in the region of the arytenoid and vocal process. As a rule endothelioma does not produce hoarseness: the patient usually complains of vocal fatigue and discomfort. This tumor is potentially malignant and recurs readily if not removed properly. The small mass must be excised wide of its base and then cauterized by means of coagulation diathermy. The tumor was removed in one of my cases and the base coagulated four years ago. To date there has been no recurrence.

Cysts of the larynx are relatively rare and when present are frequently found either on the epiglottis or the vocal cords. They may, however, be found present in the ventricles, arytenoids, aryepiglottic folds or ventricular bands. The most common type of cyst encountered in the larynx is the retention cyst. Myerson in an excellent article mentions three other types, the embryonal or congenital, the lymph or blood and the traumatic or implantation cysts. Two cases have recently come under my observation, both belonging to the retention cyst types and both occurring on the vocal cords. Suspension laryngoscopy affords an ideal surgical approach for such tumors. Two-handed surgery facilitates the accurate extirpation of the cysts.

Malignant tumors occur within the larynx with such relative frequency that we must be on our constant guard for such lesions. MacKenty stated that malignant tumor of the larynx accounts for 5 per cent of all malignancies.

From a surgical and prognostic standpoint, the exact location of the malignant tumor is of the utmost importance. The classification of these lesions into the intrinsic and extrinsic types is likewise of extreme surgical and prognostic importance.

Early voice changes are usually the first manifestations of an intrinsic lesion. Such lesions usually are found on the anterior two thirds of the vocal cords, are slow in growth and extend in the longitudinal plane of the cord with subsequent infiltration of the surrounding tissue. In the early case, carcinoma of the larynx is amenable to endolaryngeal surgery or to laryngofissure. When extension and infiltration has occurred more radical surgery must be employed. Unfortunately all carcinomas of the larynx do not arise from the vocal cords, consequently we do not always have that

symptom of hoarseness as a warning signal of danger. Extrinsic cancer of the larynx seldom gives warning of its presence until well advanced. The location of the lesion may be on the ventricular band, aryepiglottic fold or epiglottis and in the early stages there is no pain or impairment of the voice. The prognosis in these cases depends entirely upon the extent of the lesion. The advent of radiation in conjunction with surgery offers to these patients greater chances for a cure. The surprising and amazing results obtained in occasional hopeless cases with radiation stimulates the hope that the future holds more in store for these unfortunate victims.

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SUPPURATIVE PERICARDITIS

PERICARDITIS, unlike other phases of cardiac surgery, has long been under the ken of physicians: its treatment is new only in method. The main difficulty has always lain in the diagnosis. With great regret it must be admitted that too few correct diagnoses of pericarditis in its early stages are made even yet.

Galen recognized pericarditis in animals and suspected the existence in man. Rondelet described the pain, dyspnea and syncope of pericarditis, and Riolan, in 1649, suggested incision and drainage. One hundred years later Senas described pericarditis and proposed incision and drainage through the intercostal space to the left of the sternum. Morgagni, in 1761, described the great danger of effusion into the pericardial cavity due to compression of the heart. Then one wanders through a period of doubt and uncertainty in regard to both diagnosis and treatment with such men as Auenbrugger, Corvisart and Laennec unable to come to any definite conclusion mainly because of the difficulty of making a correct diagnosis but partly because of the fear of injury to the heart.

Romero in 1819 performed the first successful pericardiotomy and Hillsman in 1844 operated on the first case of suppurative pericarditis. These attempts were all more or less stabs in the dark because the physiology and pathology of the condition were not well established. Rose, in 1884, with his description of tamponade of the heart and Cohnheim, in 1889, with his physiologic experiments gave a sound basis for treatment of pericardial effusion. This

work has been clarified in the past decade by the experimental work of Beck, Ochsner, Shipley and others. These experiments have laid the technical basis for future work.

These have shown that the rapidity with which the pericardial sac is filled with either blood or serous effusion is an important factor. Beck states that in experiments on dogs the injection of 150 c.c. of fluid into the pericardial sac of a dog over a period of thirty minutes is fatal. Clinically, however, the formation of an effusion over a long period of time—as much as two to four liters—has been found in the pericardial sac before symptoms of heart tamponade were noticed. Here we may go back to the experiment first performed by Cohnheim. Cohnheim proved that as the pressure in the pericardial sac increases there is increasing obstruction to the filling of the heart, with a consequent fall in arterial pressure and rise in venous pressure. The same effects are produced by subjecting the heart to atmospheric pressure.

Only some two hundred operative cases have been reported in the literature but autopsy statistics prove that suppurative pericarditis is not rare. It is indeed found in more than 10 per cent of all fatal cases of pneumonia. It is also frequently associated with septicemia, rheumatic fever and osteomyelitis. Pneumococcus can be isolated from about half the cases; streptococcus and staphylococcus account for most of the rest.

The symptoms of suppurative pericarditis are those of sepsis plus those of embarrassed circulation; precordial pain is usually present. As Osler said, "One should always have the condition in mind when a patient with pneumonia or septicemia does not do well and frequent examination should be made. Diagnosis should not be more difficult than in encapsulated pneumonia." The patient is acutely ill. Dyspnea and shortness of breath vary with the degree of venous congestion. The heart sounds are rapid and usually muffled, though at times they seem accentuated as if the heart were floating in the effusion and so in immediate contact with the chest wall. The apex beat is weak and indistinct. At some time a friction rub may be present. The precordial dulness varies with the size of the effusion. Posteriorly there is dulness over the mediastinum. Electrocardiographic tracings are of little value. X-ray examinations, on the other hand, should always be made as they are almost diagnostic. If pus can be aspirated through a large needle, it is reassuring to the surgeon who plans to operate at once. However, pus can easily be missed and exploration of the pericardial cavity with a needle is not without danger. Paracentesis pericardii is therefore of doubtful value in diagnosis.

Although suppurative pericarditis has been recognized from almost the beginning of medicine, the actual constructive clinical work has taken place within the last twenty-five years. A great deal of credit is due to the surgeons in the Southeast. Notable contributions by Matas, Winslow and Shipley, and Bunch have gone a long way to bring this subject to the attention of the average physician.

The mortality of suppurative pericarditis treated medically is 100 per cent. Truesdale found a mortality of 42 per cent in 152 operative cases assembled from the literature in 1933. Two-thirds of these patients were from 5 to 30 years of age.

Adequate dependent drainage by open operation as soon as the diagnosis is made regardless of how moribund the patient may appear is the only safe method of treatment. The operation is a simple procedure associated with little or no shock and usually done under local anesthesia. The approach to the pericardium is made through the so-called triangle of safety or uncovered triangle of Voinitsch. This is behind the sternum and extends irregularly to the left. The apex is above at about the level of the fourth costal cartilage and the base below at the level of the base of the xiphoid.

Shipley also feels that it is wise to explore the pericardial cavity for walled-off pockets of pus and adhesions. This is done very gently with the finger with as little trauma and as little pull on the atriums as possible. He refrains from irrigating the cavity afterwards. Adhesions seem to form around the opening whether drains are used or not, but most surgeons use drains.

Empyema is the most common complication though metastatic abscess in other parts of the body sometimes kill. The patient's strength and resistance have a direct bearing on the mortality.

Paracentesis, it must be emphasized, is utterly inadequate in the treatment of supperative pericarditis.

To summarize in the words of Bunch:

1. Suppurative pericarditis is not rare.
2. It is a disease of young adults and children.
3. Although marked by characteristic physical and roentgen findings, it is too infrequently diagnosed clinically.
4. The condition is not diagnosed because it is not suspected and is not looked for.
5. Cases treated medically have a mortality of 100 per cent.
6. Treatment is exclusively surgical.
7. Prompt pericardiotomy should materially reduce the present surgical mortality of 40 percent.
8. Close cooperation of clinician, roentgenologist and surgeon at all times is essential.

BOOK REVIEWS

The Editors of THE SOUTHERN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

A TEXTBOOK OF SURGERY. By American Authors. Edited by FREDERICK CHRISTOPHER, B. S. M. D., F. A. C. S., Associate Professor of Surgery at Northwestern University Medical School; Chief Surgeon, Evanston (Illinois) Hospital. 1608 pages with 1349 illustrations on 730 figures. Price $10. Philadelphia and London: W. B. Saunders Company, 1936.

To cover every field of surgery today, brain tumors and ingrowing toenails, cancers of the stomach and pruritus ani, fractures of the femur and infected tonsils, cleft palate and urethral stricture, tumors of the womb and sebaceous cysts, all in one volume was indeed an ambitious undertaking. This undertaking has been successfully accomplished in a massive tome, the product of 184 surgeons: "with few exceptions these men are actively engaged in teaching surgery to medical students." Nineteen of the authors have contributed to THE SOUTHERN SURGEON and several of the many illustrations were first published here. Dr. Christopher has done a remarkably fine job of keeping his many authors within the space limitations, and he has added notes here and there in amplification or modification of some of their opinions. These notes, some of which are particularly appealing, serve to bind the many sections into a unified whole. The book is well balanced: seven lines are devoted to laryngectomy, but fifty-two pages to tumors of the breast.

This book contains everything the undergraduate could be called upon to know (the undergraduate however should have mature guidance in emphasis and selection). Although this work could hardly make a finished surgeon of a young doctor, there is no way except through experience in assisting competent men and in doing some surgery under their supervision that a boy can be converted into a surgeon. It is doubtful too, whether an average surgeon should attempt to do a complicated operation the like of which he has never attempted before without at least some personal observation, and therefore the reviewer does not think that the book is too greatly lacking in detail. On the other hand, it is so full and so up to date (it includes even Beck's muscle graft to the heart) that it will serve as a condensed system of surgery at a fraction of the cost of a many-volume system: it can thus be discarded without great loss when a second edition appears.

CLINICAL MISCELLANY: VOL. 2, 1935. FRANCES F. HARRISON, M. D., CHARLES C. MCCOY, M. D., MONROE A. MCIVER, M. D., GEORGE M. MACKENZIE, M. D., MARJORIE F. MURRAY, M. D., ROBERT M. PIKE, Ph. D., JOHN H. POWERS, M. D., DOROTHY K. SCHEIDELL, M. D., RADFORD C. TANZER, M. D. 218 pages with 50 illustrations. Price, $3. Springfield and Baltimore: Charles C Thomas, 1935.

Rochester, Minn., now boasts of more than 20,000 inhabitants. Halstead, Kansas, with less than 1,500, also boasts of a surgical genius. But geniuses are as rare in surgery as in other lines. It is interesting therefore to read of the kind of medicine that is being practiced today in another small town. Cooperstown, New York, does not claim 3,000 souls, but it is the home of the Mary

Imogene Bassett Memorial Hospital. For the second year the Staff of this Hospital has published a book to record their most interesting observations.

"The insight, humanitarianism and art of traditional medicine is slowly being integrated with what science, experiment and precise chemical and physical technics have to contribute towards a better comprehension of the endlessly complex phenomena of disease." Underlying the book is propaganda for further integration. Again the contributors have produced an interesting volume dealing with conditions that the physician in the smaller communities is apt to encounter. This book should prove particularly stimulating to such men as evidence that nowadays the most scientific medicine can be practiced in small places. The art of medicine has always been well practiced there.

Mr. Thomas has, as usual, given the book a handsome appearance.

SURGICAL CLINICS OF NORTH AMERICA. Issued serially, one number every other month. Volume 16, Number 1. Chicago Number—February, 1936. 356 pages with 78 illustrations. Per Clinic year February, 1936 to December, 1936. Paper, $12.00; Cloth, $16.00 net. Philadelphia and London: W. B. Saunders Company, 1936.

It is a pleasure to take up a copy of the "Surgical Clinics of North America." These cloth-bound volumes which come out every second month are organized like a brief postgraduate course in surgery. The convenient size, good paper and type add to their attractiveness. If this particular copy is a sample, startling advances in surgery are not announced in its pages, but material which has proved its worth is presented in an informal style which makes it almost as well worthwhile as actually listening to the surgeon and seeing his clinical material.

This number opens with a symposium on "Cancer of the Cervix" which is splendidly done, although the reviewer notes that one author considers the Schiller test worthless and another believes it valuable. Dr. Bevan's discussion of appendicitis is a contribution of great value, both historically and practically. Not every surgeon will be greatly interested in every paper, but every surgeon will find several that will interest him and some that will benefit him. If he follows this series carefully, he should never fall behind in the procession.

PARENTERAL THERAPY. A Ready Reference Manual of Extra-Oral Medication for Physicians, Dentists, Pharmacists, Chemists, Biologists, Nurses, Medical Students and Veterinarians. By WALTON FOREST DUTTON, M.D. Formerly Medical Director, Polyclinic and Medico-Chirurgical Hospitals Graduate School of Medicine, University of Pennsylvania, Visiting Physician to the Northwest Texas Hospital, Colonel, M. O. R. C., etc. and GEORGE BURT LAKE, M.D., formerly Special Lecturer in Hygiene, Purdue University, Editor, Clinical Medicine and Surgery, Colonel, M. O. R. C., etc. 386 pages with 90 half-tone illustrations and line engravings. Price $7.50. Springfield and Baltimore: Charles C Thomas, 1936.

Though major surgery cannot be learned from a book, parenteral therapy can. This new Thomas book is a masterpiece of detail and illustration. It supplies a great many little bits of information that one may have picked up through bitter experience or that one may have been expected to know but somehow doesn't and was ashamed to ask about. It includes a therapeutic index of 30 pages and 143 pages of pharmacologic notes. The reviewer, however, must register an emphatic kick against some of the pharmaceuticals that

have been included. The authors, perhaps in their zeal to bring the book right up to the minute, have included many items that have not been accepted by the Council on Pharmacy and Chemistry of the American Medical Association; some of these, it would appear, were included on nothing more substantial than the manufacturer's claims, and the claims of some of the manufacturers represented are not worth very much. Certainly their advertisements would not be accepted by the *Journal of the American Medical Association* nor by THE SOUTHERN SURGEON.

This book will have an enormous sale.

NEW PATHWAYS FOR CHILDREN WITH CEREBRAL PALSY. By GLADYS GAGE ROGERS, Director of Robin Hood's Barn, a Camp School for Children with Cerebral Palsy, and LEAH C. THOMAS, Director of Therapeutics at Robin Hood's Barn; formerly Associate Professor of Hygiene and Physical Education, Smith College, and Director of Corrective Exercises under the Direction of Dr. Joel E. Golthwait and Dr. Robert B. Osgood, Boston, Mass. 167 pages with 11 illustrations, New York: The Macmillan Company, 1935.

This book is not only delightful reading but it also is an impressive exposition of what can be done for these afflicted children that we have been prone to consider hopeless.

The Southern Surgeon, the second regional journal devoted to a specialty in the United States, is published by The Southern Surgeon Publishing Company, a subsidiary of The Southeastern Surgical Congress, for the advancement of surgery particularly in the South. In addition to publishing papers irrespective of their origin, it aspires to encourage surgeons in the Southern States to record their own observations and original work.

Manuscripts for publication, books for review, and correspondence relating to the editorial management should be sent to the Editor-in-Chief, Dr. L. Minor Blackford, 104 Ponce de Leon Ave., N. E., Atlanta, or to one of the other editors. Other communications should be addressed to Dr. B. T. Beasley, 478 Peachtree Street, N. E., Atlanta.

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References should conform to the style of the Quarterly Cumulative Index Medicus, published by the American Medical Association. This requires, in order given: name of author, title of article, name of periodical, with volume, page, month (day of month if weekly) and year.

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